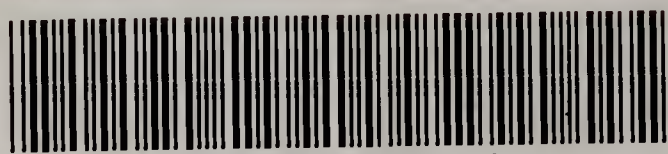


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FIG. 1.—The Hospital of the Protestant Episcopal Church in Philadelphia.



HOSPITAL

OF

THE PROTESTANT EPISCOPAL CHURCH
IN PHILADELPHIA

MEDICAL AND SURGICAL
REPORTS

OF THE

EPISCOPAL HOSPITAL

VOLUME I

PHILADELPHIA
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The Medical Staff desire to express their high appreciation of the liberality of the Board of Managers of the Hospital in making provision for the publication of this Volume.

THE Committee of Publication thinks it proper to say that the Hospital holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in this volume.

FRANCIS W. SINKLER, M.D.,
ASTLEY P. C. ASHHURST, M.D.,
COURTLAND Y. WHITE, M.D., *Chairman.*

This volume has been edited by
ASTLEY P. C. ASHHURST, M.D.



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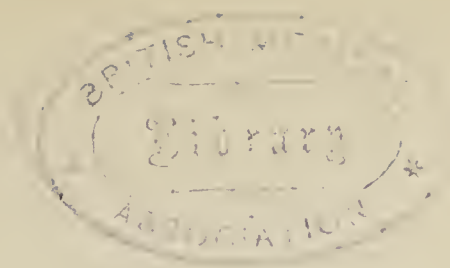
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FIG. 2.—Administration Building.



BOARD OF MANAGERS
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Persons having communications to make to the Board, will address the Secretary as above.

For information concerning the Hospital (patients, admissions, etc.), see or address the Superintendent, at the Hospital, Front Street and Lehigh Avenue.

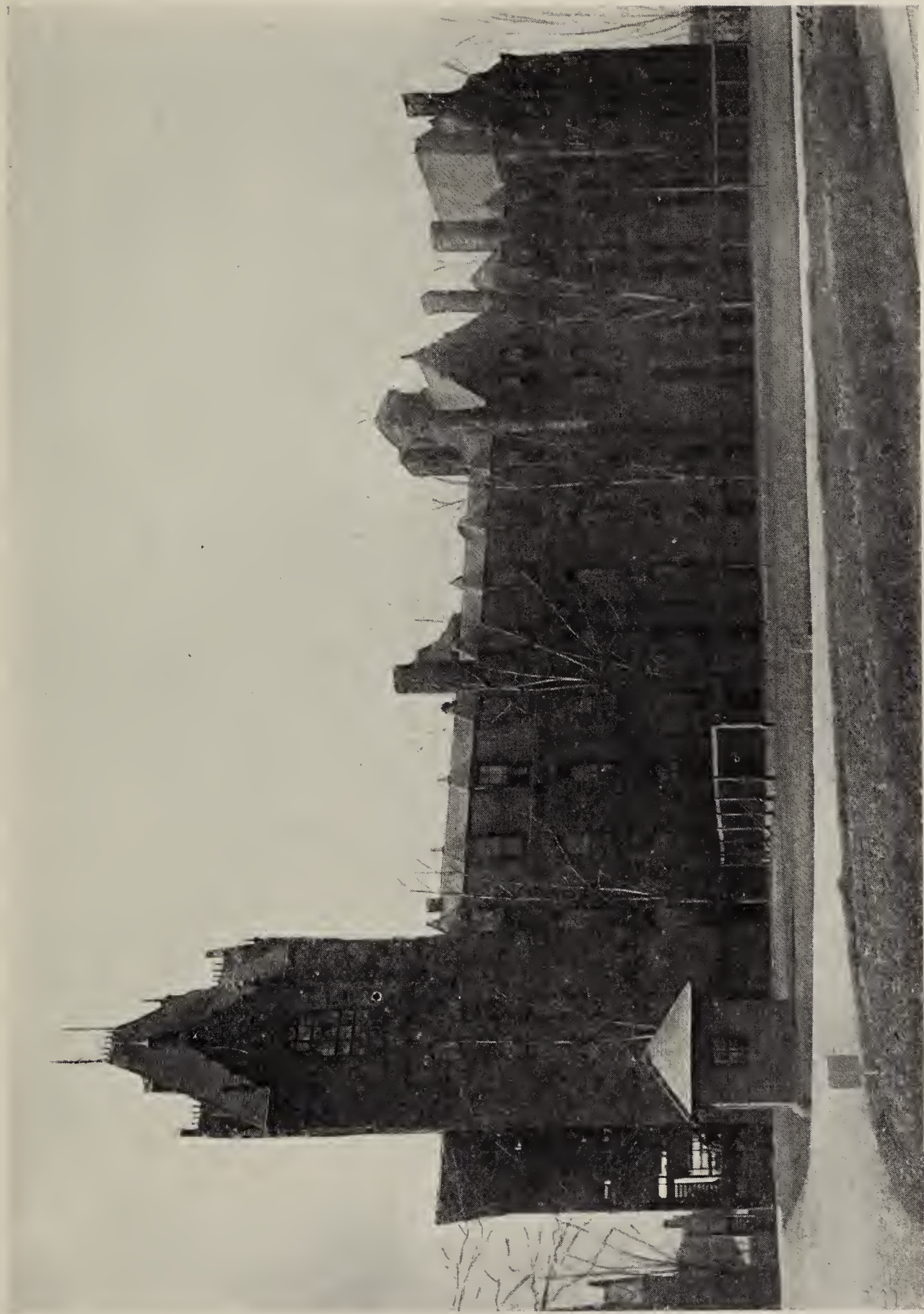


FIG. 3.—Harrison Memorial House for Incurables.

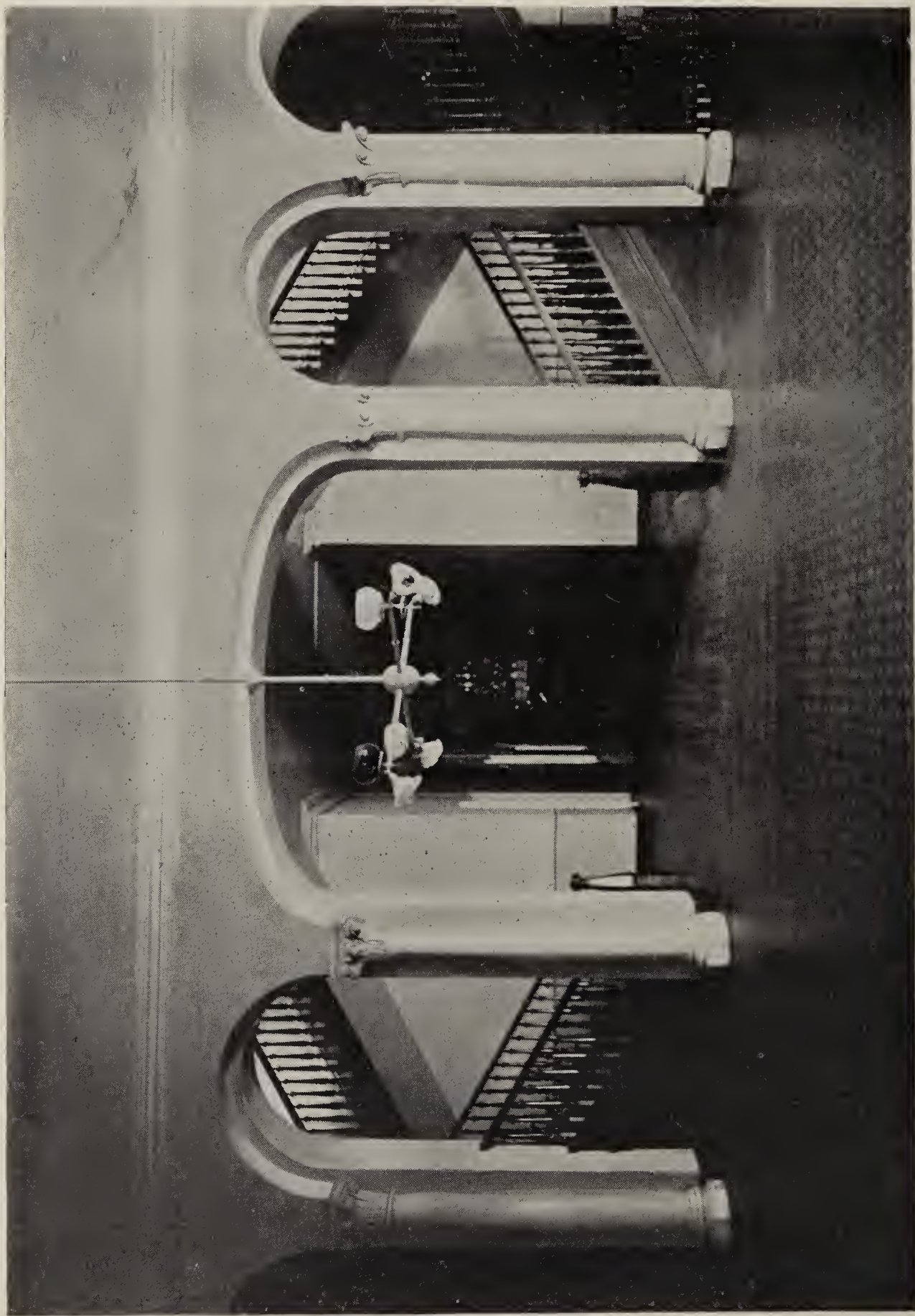


FIG. 4.—Main Stairway.

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GEORGE H. ENGLISH, PH.G.

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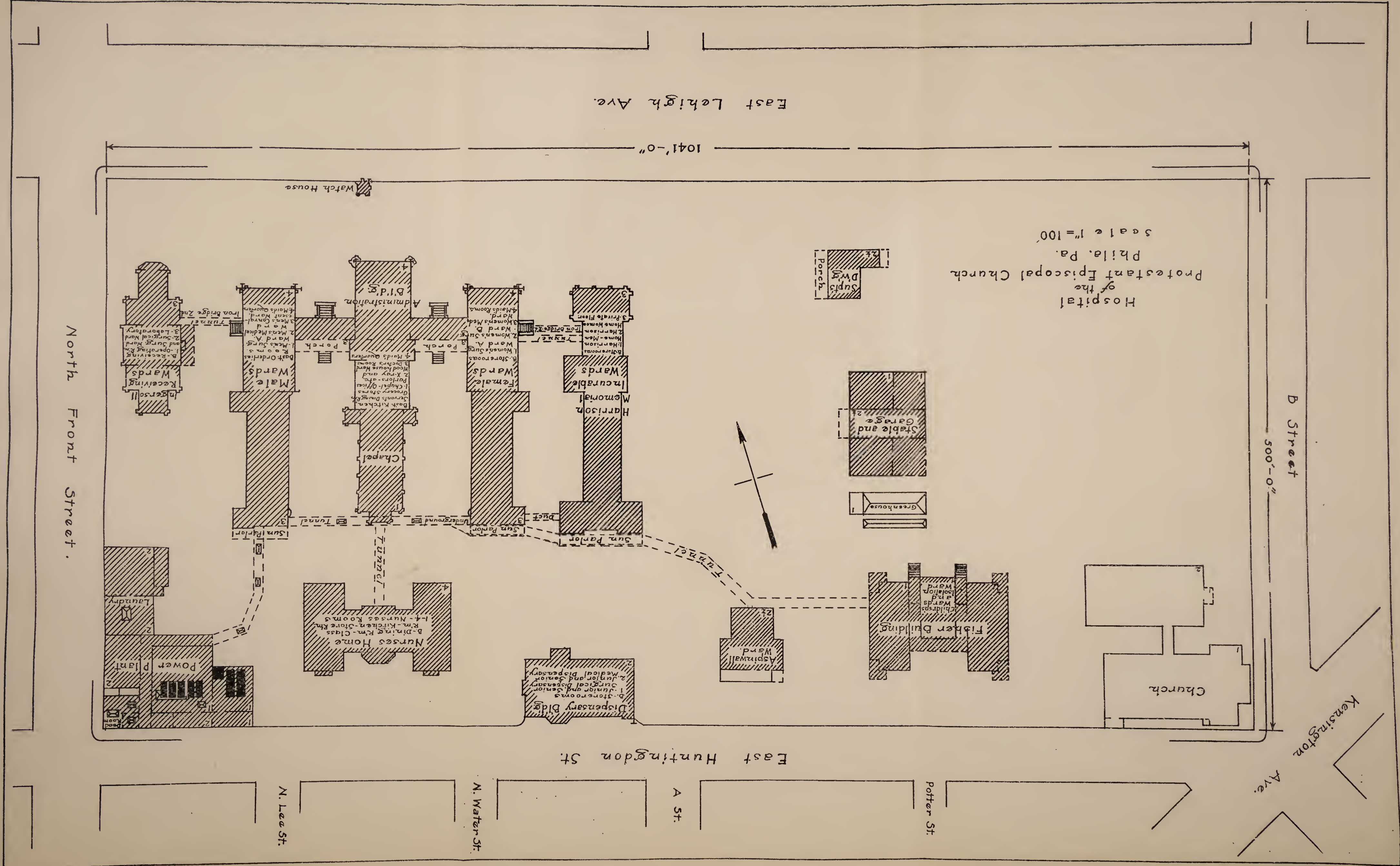
Radiographer to the Hospital; Radiographer to St. Agnes's Hospital; Physician to the State Hospital for the Insane, Wernersville, Penna.

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PAPERS BASED ON WORK DONE IN THE EPISCOPAL HOSPITAL, 1912

FROM THE MEDICAL DEPARTMENT OF THE HOSPITAL

From Dr. Stevens' Service.

Stevens: Malignant Disease of the Lung, with Special Reference to Sarcoma.

From Dr. Sinkler's Service.

Sinkler: Some Cases of Neuritis and Neuralgia of Varied Type and Causation.

From Dr. Norris' Service.

Pemberton: A Case of Acute Suppurative Peritonitis of Unknown Origin, Simulating Meningitis.

From Dr. Piersol's Service.

Piersol: The Clinical Significance of Extreme Degrees of High Blood Pressure, with Remarks on its Management.

FROM THE SURGICAL DEPARTMENT OF THE HOSPITAL

From Dr. Neilson's Service.

Neilson: Two Cases of Ruptured Extra-uterine Pregnancy, One of Them in an Instance of Uterus Bicornis.

From Dr. Deaver's Service.

Deaver: The Uncertainty in Diagnosis of Upper Abdominal Diseases and Conditions.

Alexander: Fracture of the Patella, with a Report of Fifty-six Cases.

Alexander: Fracture of the Radius Above the Attachment of the Pronator Quadratus Muscle.

From Dr. Frazier's Service.

Frazier: The Surgical Clinic of the Protestant Episcopal Hospital of Philadelphia.

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Ashhurst: Arthrotomy of the Knee.

Ashhurst: The Treatment of Dislocation of the Head of the Radius, Complicated by Fracture of the Ulna.

From Dr. Frazier's Service.

Ashhurst: Operative Treatment of Old Fractures of the Elbow.

Ashhurst: A Case of Bone Cyst Involving the Upper End of the Humerus.

Happel: Report of a Case of Herpes Zoster Complicated by Bell's Palsy.

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Goldberg: Choroideremia; Report of Two Cases in One Family.

From Dr. Krauss' Service.

Krauss: Pseudoglioma in Children.

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Eves: Some Causes of Cough Often Overlooked, Pertaining to the Ear, Nose, and Throat.

Eves: Enucleation of the Tonsils and Removal of Adenoids and of the Lingual Tonsil by Simple Methods.

FROM THE PATHOLOGICAL DEPARTMENT

White: Sarcoma of the Appendix Vermiformis.

FROM THE OBSTETRICAL SERVICE

Parke: Puerperal Eclampsia.

Parke: Remarks on the Treatment of Puerperal Septic Infection and Report of a Case of Septic Thrombophlebitis of Long Duration.



FIG. 5.—The Aspinwall Building for Convalescent Children.



FIG. 6.—Fisher Building (Children's Hospital).



FIG. 7.—Solarium in Children's Ward.



FIG. 8.—Operating Room in the Fisher Building (Children's Hospital).



FIG. 9.—Ambulance Garage.

THE EPISCOPAL HOSPITAL

By EDWARDS F. LEIPER, COMMANDER U. S. N., RETIRED
SUPERINTENDENT OF THE HOSPITAL

THE Hospital of the Protestant Episcopal Church in Philadelphia was chartered July 18, 1851. At that time (aside from the Philadelphia Hospital, which was sustained as an almshouse from public funds) the Pennsylvania Hospital, established for a century at Eighth and Pine Streets, was the only general hospital in Philadelphia, which then had a population of more than four hundred thousand. Besides the two hospitals mentioned above there were also four dispensaries and the Wills Hospital, which was quite small, and was devoted to the care of the blind and lame. There were but little more than one hundred beds in the existing hospitals for the accommodation of the sick poor. Viewing the situation from the standpoint of today the necessity for greater provision for the care of the sick was very evident. Impressed by this necessity a number of public spirited and benevolently inclined persons in response to inquiries of the Right Reverend Alonzo Potter (then Bishop of the Diocese of Pennsylvania), as to the needs of the Diocese, pointed out to him the need of a hospital for the care of the sick, in which the comforts of instruction in religion might be supplied. The following resolutions were adopted at the First General Meeting of the Clergy and Laity, March 14, 1851.

On motion of the Hon. Jos. R. Ingersoll, seconded by Mr. George M. Wharton.

Resolved, That in the opinion of this meeting an effort ought to be made to enlarge the hospital accommodations in this City and County of Philadelphia for the destitute sick.

Resolved, That in the proposed institution the instructions and consolations of religion ought to be regularly supplied to the inmates and attendants, and that to secure this end effectually and in a way most conducive to Christian Charity it is expedient that the effort be made under the auspices of Protestant Episcopalians.

Resolved, That a committee of nine persons be appointed to consider and report to a future meeting the most eligible plan for the attainment of the object proposed.

The Committee consisted of—

Rev. Dr. Howe,	Dr. Wm. Keith,
Rev. Mr. Odenheimer,	Mr. J. Fisher Leaming,
Hon. J. R. Ingersoll,	Mr. G. M. Wharton,
Dr. Caspar Morris,	Mr. Wm. Welsh,
Mr. Peter McCall.	

To which were added Bishop Potter and Mr. John Welsh, Jr.

In his efforts to secure the establishment of the hospital, Bishop Potter was ably assisted by the late Dr. Caspar Morris, who issued an appeal on behalf of the sick, which was largely circulated and splendidly endorsed by the clergy and had great influence in attracting the interest of the laity in sending generous contributions. Two ladies, Miss Ann Leamy and her sister, Mrs. Elizabeth H. L. Stout, presented to the managers as a site for the hospital a block of land at Front Street and Lehigh Avenue, on which stood their family home. At that time this was far in the country and many were of the opinion that it was too far from the city to be of service. However, the gift was accepted and on December 11, 1852, the hospital was formally opened in the Leamy mansion, and from that date until the end of the year six patients had been admitted. Such was the beginning of the hospital of the Protestant Episcopal Church in Philadelphia, popularly known as the "Episcopal Hospital."

With the opening of the institution, the real work of the founders was but just begun; and during the next eight years, while a fund was being collected, with which to build a hospital

commensurate with the needs of the city, patients were treated in the Leamy mansion, where twenty-three beds had been installed.

The corporate title of the hospital is "The Hospital of the Protestant Episcopal Church in Philadelphia." Its objects, as provided by the original charter are:

1. To provide medical and surgical aid and nursing for the sick and disabled, either in the wards of the hospital or at their homes.

2. To instruct and train suitable persons in the duties of nursing and attending upon the sick.

3. To provide the instructions and consolations of religion according to the principles of the Protestant Episcopal Church for those who are under the care of the institution.

4. Such other purposes, incidental and kindred to those above mentioned as the Board of Managers, hereinafter to be provided, may prescribe.

The Board of Managers each year appoints the Medical Staff for the current year. The entire medical care of the patients devolves on the Medical Staff. The charter provides for the admission of all accident cases within twenty-four hours of the time the injury was received, and of other patients without discrimination as to creed, color, or country, giving preference among incurable patients to members of the Protestant Episcopal Church; and among other cases, to those considered most necessitous, urgent, and dangerous and such as are most likely to admit of being relieved. Those suffering from contagious diseases are not admitted.

In the year 1860, the number of indoor patients treated was 403, with a daily average in the house of 28; while 5000 outdoor patients were treated in the same year. The rapidly increasing number of factories in the district already had demonstrated the wisdom of the founders of the hospital in selecting its location. On May 24, 1860, the cornerstone of the main group of hospital buildings was laid by the Bishop and Assistant Bishop of Pennsylvania, with appropriate and

solemn ceremonies in the presence of the clerical and lay delegates of the Diocese then in annual convention assembled, and an immense concourse of deeply interested spectators. The buildings begun at that time still constitute the main portion of the hospital and have been the object of much admiration, because of the spaciousness of the wards, which are well lighted and ventilated, and which have continued to lend themselves admirably to the care of the sick under all changes that have occurred in medical and surgical methods of treatment. The main group of buildings originally designed consists of three parallel pavilions extending north and south, connected with each other by a transverse corridor near the north end, running two hundred and fifty-eight feet, the length of the entire front of the three original pavilions. The depth of the pavilions is about two hundred feet and the spaces between them about sixty-three feet each. The central pavilion is four stories in height; a chapel, capable of seating about three hundred and fifty persons, being at its rear end. This building is used for administrative purposes and quarters for the house staff.

The east and west buildings are three stories in height at the south end where the wards are located, and four stories at the north end, the fourth floor in each building being used as quarters for domestics. The wards are each one hundred and twenty feet in length and thirty feet in width. Each ward contains thirty beds with windows on each side which are of full width and extend to within a few inches of the ceiling, affording good ventilation and ample light. At each end of the wards and outside of the main building are towers which contain toilet rooms and bath rooms, and large water tanks near the roof. Outside the space devoted to the wards on each floor are a number of smaller rooms in which special patients may be placed when the doctors think they may be treated more beneficially out of the wards. In this part of the building, too, are located the serving kitchens and dressing rooms for surgical cases.



FIG. 10.—Main Corridor.

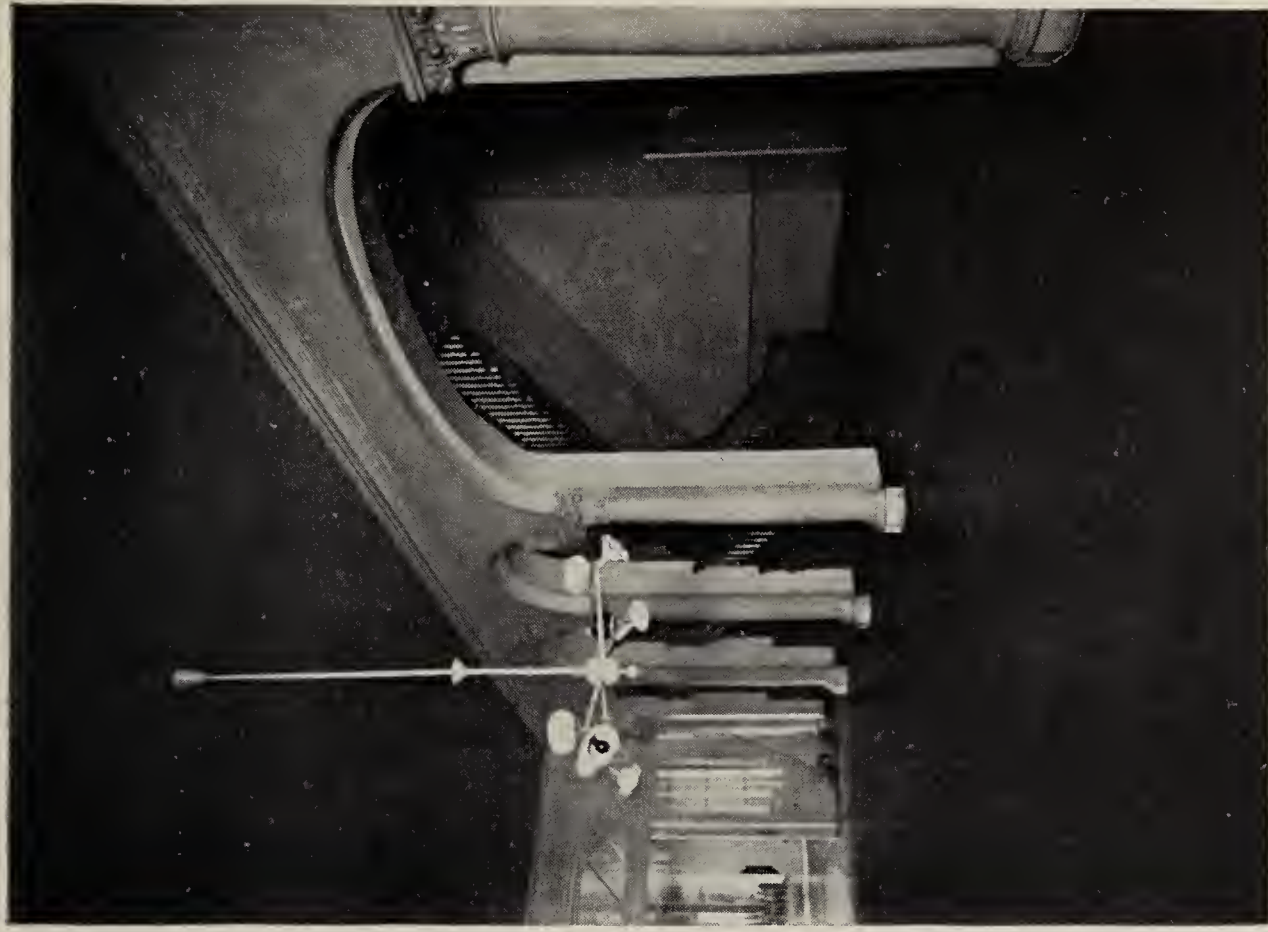


FIG. 11.—Hallway in Administration Building.

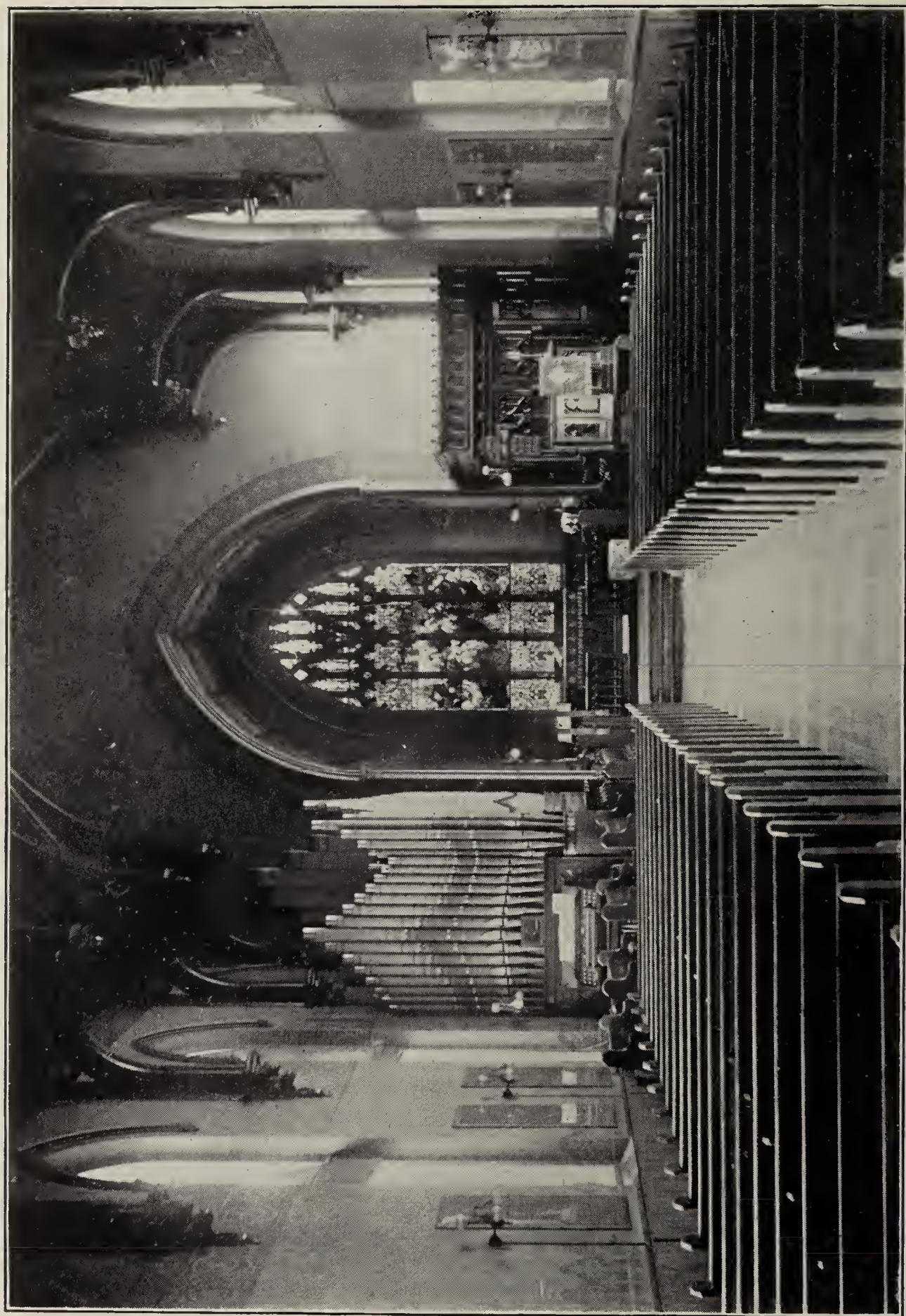


FIG. 12. --The Chapel of the Hospital.

Since the erection of this group of buildings, the central and western portions of which were begun in 1860, and the eastern wing of which was finished in 1875, and first occupied in 1878, two other pavilions have been added to the original group. One of these, added on the east side, is called the Harrison Memorial House, and contains eighty beds for incurable patients on the first and second floors, and twenty private rooms and a private ward for women on the third floor. The other pavilion was added on the west side and is called the Ingersoll Building. It contains a receiving ward, and the two main operating rooms of the hospital, with adjacent recovery rooms, two surgical wards having twelve beds each, and the Pathological Department. These additions were made in 1890 and 1894 respectively.

In 1901, the Nurses' Home was built on the south side of the Hospital grounds, by three generous benefactresses of the Hospital. This building is three stories high above the basement and contains rooms for one hundred nurses. At the present time a fourth floor is being added, which will make room for thirty additional nurses; and by a change in the form of roof, there will be added a spacious sun parlor and a very large roof garden over the entire building.

In 1904 the Aspinwall Building was erected, for the care of convalescing female white orphan children. In 1906 the Fisher Building was erected for an isolation ward; it contained four separate wards with four beds each, and an administration building. Owing to the erection of a new Municipal Hospital for the care of patients suffering from contagious diseases and new regulations established by the City authorities governing the care of such patients in hospitals, this building was used but little until the Autumn of 1911, when alterations were made adapting the greater part of it for use as a Children's Hospital. It has beds for forty-five patients and is constantly full.

The Superintendent's dwelling house, the stable and garage, power-house, laundry, machine shops, carpenter shop, and paint

shop are other buildings located on the ground apart from the buildings devoted to the care of patients. The Out-Patients' Building is in the rear of the Hospital ground, facing Huntingdon Street, and is a two-story building admirably adapted to its purpose.

The total capacity of the Hospital is five hundred beds (including two open-air tuberculosis wards of six beds each, recently opened), 85 per cent. of which are occupied almost constantly. The growth of the Hospital has been continuous since its foundation. In the latter part of 1852, six patients were admitted to the Leamy mansion. During the year 1853, the total number of the patients treated was one hundred and seventy-four. During the year 1862, the west wing and central building were completed, and immediately were put in readiness, during the month of July, to receive the first patients, an installment from the army; and soldiers continued to occupy the wards until the close of the Civil War. The new wards of the Hospital were promptly filled, so that at the end of the year 1862 there were two hundred and thirty wounded or sick soldiers in them. The completion of the chapel followed that of the other buildings very quickly. The Leamy mansion still continued to be used for the housing of patients, in what was known at that time as the Civil Department, to distinguish it from the Military Wards, in which the soldiers were treated. The total number treated in 1862 was three hundred and sixty-nine, while the last annual report shows that the total number treated in the house during the year 1912 was about five thousand, while more than twenty thousand new cases were treated in the Out-Patient Department.

The balance sheet of the Hospital shows that the hospital has assets, practically all of which represent investments in income-producing property, amounting to about \$2,500,000 not including the Hospital real estate and buildings, which cost originally upward of \$650,000. The annual income aside from legacies amounts to about \$152,000. This is de-

rived from various sources, as follows: Invested funds, \$114,000; contributions from individuals, about \$10,000; collections from Churches, about \$12,000; board of patients, \$13,000; and other sources amounting to about \$3000. The expenses of the hospital amount to about \$200,000 a year, exclusive of repairs and improvements. The Hospital receives no appropriation from the State, being supported entirely by the liberality of its many friends who have contributed to the endowment fund and who continue to assist in building up that fund.

The Hospital receives patients of all characters in general, and its doors are always open to the poor, and the vast majority are treated absolutely free. There are accommodations for a limited number of private patients, toward whom the same liberal spirit is extended as toward the free patients, the cost of room and treatment being made as little as possible. Besides the two principal departments of Medicine and Surgery, there are the usual departments found in larger hospitals—Ophthalmic, Aural and Laryngeal, Pathological, Radiographic, Orthopædic, Genito-Urinary and Dermatologic Departments.

Connected with the Hospital is a nursing department, consisting of eighty-four pupil nurses and sufficient graduate nurses to bring the total to more than one hundred. This department is about to be enlarged by the addition of twenty or thirty more pupil nurses.



FIG. 13.—Nurses' Home.

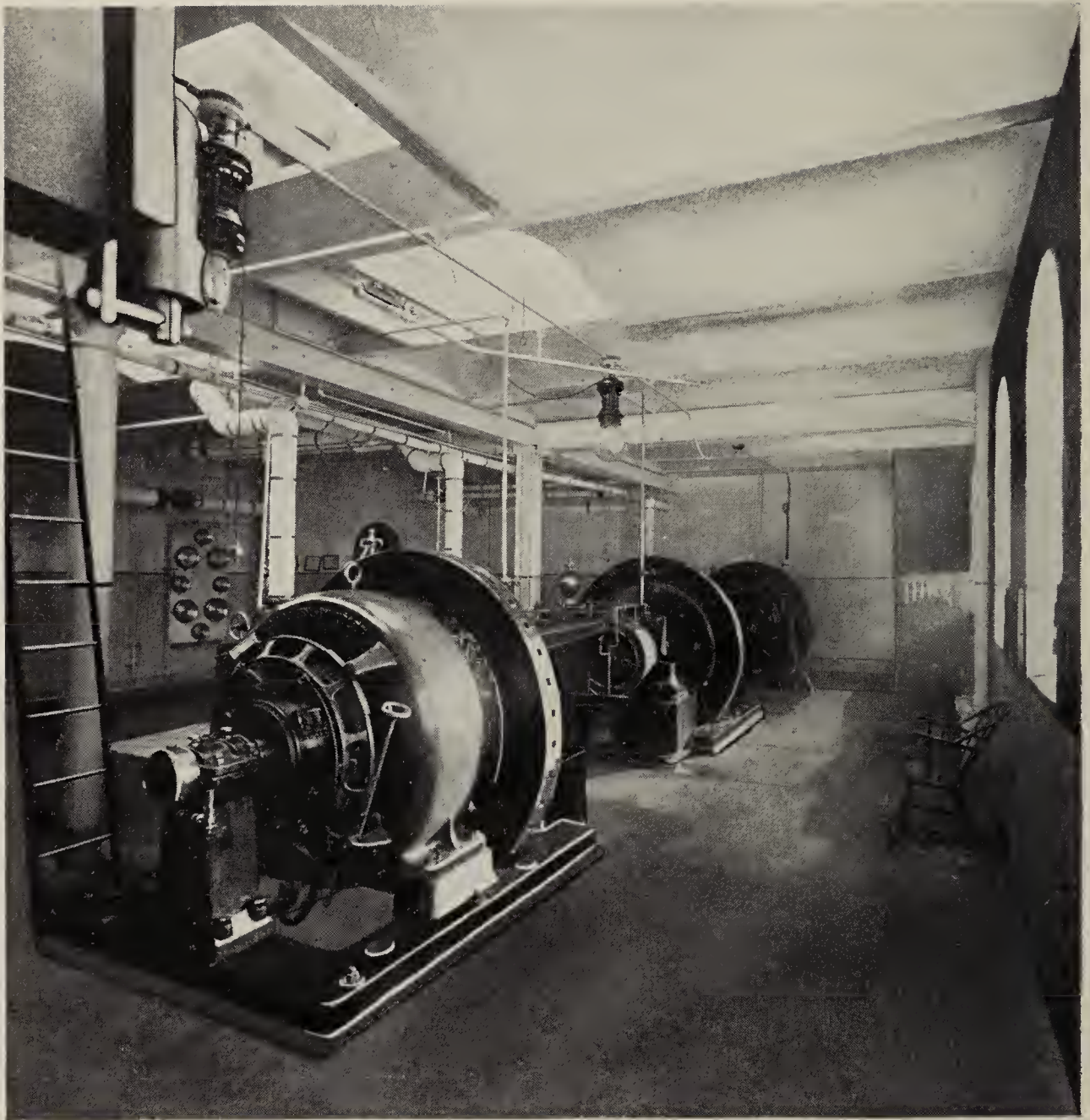


FIG. 14.—Power House, Dynamo Room.

REPORT OF SUPERINTENDENT FOR 1912.

STATISTICAL REPORT.

SUMMARY.

During the year 4433 patients have been admitted to the Hospital and 8 patients to the Harrison Memorial Home. At the date of the last report 213 patients were in the Hospital and 72 in the Harrison Home, making the total number treated during the year, 4718. Of these, 4395 have been discharged, 2742 cured, 1004 improved, 241 unimproved, whilst 418 have died, leaving 323 patients under treatment, of whom 104 are medical, 147 are surgical, 72 incurable cases in the Harrison Home (140 men, 150 women and 33 children), in the wards at the end of the year.

There have been 22,755 new patients treated at the Dispensaries during the year. Of this number, 6071 have been medical, 9490 have been surgical, 4618 have been eye cases, while in the ear, nose and throat dispensary there have been 2576. The daily average of Dispensary patients has been 290. The Emergency, Receiving Ward treated 7346 patients from January 1st, 1912, to December 31st, 1912. The daily average of patients treated in the Emergency Department has been 20.

Number of days under treatment of patients who were admitted during the year 1912..... 84,151

Number of days under treatment (this year) of patients remaining from 1911..... 38,383

Total..... 122,534

Average daily number of patients in the Hospital..... 334.8

Average duration of treatment of all the patients..... 26 days.

Average duration of treatment, not including those in the Memorial House..... 20 $\frac{1}{3}$ days.

Cost of patients (per day)..... \$1.49

Average daily number of patients in the Dispensaries..... 290

Average daily number of patients in Emergency Department 20

Average daily number of prescriptions..... 77

The number of visits made by old and new patients to the Dispensaries during the year..... 88,995

TABULAR STATEMENT OF NUMBER OF PATIENTS

ADMITTED TO THE PROTESTANT EPISCOPAL HOSPITAL DURING SIXTY-ONE
(61) YEARS, TOGETHER WITH THE LENGTH OF TIME UNDER TREATMENT,
TOTAL COST, COST PER DAY, AVERAGE DAILY NUMBER, AND THE RESULTS.

Year.	Total cost of treatment.	Patients remaining on the first day of each year.	New patients.	Total treated.	Average number of days under treatment.	Average daily number of cases under treatment.	Cost per day.	Condition when discharged from Hospital.				Total discharged.	Patients remaining under treatment on the last day of each year.
								Cured.	Improved.	No improvement.	Died.		
1852	6	6	4	2	6
1853	\$3,020 40	174	174	31	99	25	10	22	156	18
1854	3,887 47	18	271	289	25	174	41	20	22	257	32
1855	5,315 85	32	281	313	30	25	194	41	43	17	295	18
1856	6,604 19	18	358	376	32	26	\$0 58	215	60	43	30	348	28
1857	6,572 78	28	310	338	35	30	52	203	60	16	20	308	30
1858	5,070 88	30	342	372	29	27	50	270	41	16	22	340	23
1859	4,955 14	23	364	387	22	26	46	291	25	7	33	356	31
1860	4,777 76	31	372	403	27	28	50	252	72	14	30	368	35
1861	4,751 28	35	361	396	26	28	50	224	91	20	34	369	27
1862	15,911 33	27	342	369	27	26	65	217	73	26	31	347	22
1863	22,438 80	22	464	486	28	46	60	296	65	21	44	426	60
1864	20,041 33	60	592	652	43	65	75	392	83	38	66	579	73
1865	26,662 67	73	729	802	39	86	75	457	175	29	62	723	79
1866	29,147 05	79	834	913	32	82	80	579	148	26	65	818	95
1867	31,997 03	95	815	910	45	93	I 00	586	174	12	58	830	80
1868	32,281 09	80	925	1005	39	98	I 00	617	187	27	71	902	103
1869	36,912 94	103	937	1040	45	104	I 00	680	149	25	86	940	100
1870	35,096 57	100	860	960	40	105	I 00	622	156	13	68	859	101
1871	37,214 28	101	1002	1103	37	104	I 05	687	182	47	83	999	104
1872	41,337 90	104	940	1044	38	104	I 09	643	167	57	74	941	104
1873	46,408 38	104	1033	1137	39	117	I 07	611	279	49	85	1024	113
1874	46,646 38	113	1071	1184	41	118	I 07	634	302	46	87	1069	115
1875	48,138 17	115	1078	1193	42	119	I 08	765	213	18	81	1077	116
1876	45,828 70	116	1301	1417	34	120	I 06	945	270	9	88	1312	105
1877	41,108 10	105	1282	1387	38	119	95	800	389	19	71	1279	101
1878	39,386 32	101	1203	1311	38	117	89	660	424	36	90	1210	101
1879	41,749 48	101	1100	1201	41	108	91	547	330	40	92	1009	91
1880	42,395 79	91	1202	1293	33	109	90	798	239	38	113	1188	105
1881	48,164 51	105	1225	1330	47	123	I 06	849	214	38	105	1206	124
1882	54,295 08	124	1239	1363	43	144	I 04	811	223	61	130	1225	138
1883	57,415 86	138	1570	1708	33	146	I 05	990	362	64	158	1574	134
1884	56,787 98	134	1593	1727	32	148	I 03	986	350	88	143	1567	160
1885	58,156 66	160	1739	1899	31½	163	98	1007	488	84	147	1726	173
1886	58,606 02	173	1609	1782	34	166	96	1046	322	95	160	1623	159
1887	60,023 93	159	1809	1968	35	160	96¾	1242	340	52	159	1793	175
1888	63,703 24	175	1823	1998	35	172	I 00	1337	293	36	156	1822	176
1889	63,680 36	176	1791	1967	33	177	I 00	1307	305	33	145	1790	177
1890	66,995 20	177	1747	1924	34	175	I 03	1261	321	28	172	1782	142
1891	76,333 45	142	1860	2002	34	176	I 05	1191	419	44	176	1830	172
1892	84,323 84	172	2022	2194	34	207	I 11	1353	403	60	167	1983	211
1893	88,846 64	211	2109	2320	34	211	I 11	1235	522	108	217	2082	238
1894	87,776 77	238	2267	2505	32	220	I 09¼	1380	564	123	228	2295	210
1895	93,341 87	210	2490	2700	30	225	I 13¼	1579	555	96	238	2468	232
1896	93,740 37	232	2573	2805	27	233	I 09¾	1839	436	70	230	2575	230
1897	97,591 83	271	2612	2842	27	251	I 06½	1694	532	127	218	2571	271
1898	109,529 48	271	2764	3035	27½	286	I 04¾	1977	415	83	271	2746	289
1899	105,218 91	289	2737	3026	23¾	259	I 11¼	2024	361	115	276	2776	250
1900	111,838 78	250	2936	3186	22¾	264	I 16½	1971	516	120	327	2934	252
1901	109,077 87	252	2720	3081	22¾	253	I 18	1815	546	92	274	2727	254
1902	112,936 07	254	2750	3004	24	263	I 17½	1710	647	122	275	2754	250
1903	124,393 24	250	2888	3138	24	273	I 25	1765	716	67	314	2862	276
1904	133,757 53	276	2951	3227	24	279	I 30	1804	719	144	296	2963	264
1905	136,163 59	264	3442	3706	23½	303	I 23	2313	677	123	303	3416	290
1906	144,704 16	290	3936	4226	20⅞	307	I 29	2717	773	101	340	3931	295
1907	153,512 57	295	3483	3778	21½	286	I 47	2503	541	123	369	3536	242
1908	155,245 59	242	3256	3498	22½	280	I 51	2203	612	143	281	3239	259
1909	152,344 95	259	3461	3720	20¾	280½	I 48¾	2602	472	66	299	3439	281
1910	155,054 66	281	3699	3980	19	277⅞	I 53	2417	873	120	328	3738	242
1911	175,913 10	242	4015	4257	19½	298½	I 62	2520	951	161	340	3972	285
1912	183,050 29	285	4433	4718	20⅓	334.8	I 49	2742	1004	241	408	4395	323

To this may be added the following tabular exhibit of the general expense account for the last forty years:—

YEAR.	MEDICINES.	HOUSEHOLD.	IMPROVEMENTS AND REPAIRS.	TOTAL.
1873	\$6,217 12	\$40,541 18	\$1,584 46	\$48,342 76
1874	6,007 78	40,638 60	2,132 33	48,778 71
1875	7,294 50	40,894 34	1,683 60	49,872 44
1876	5,942 54	40,325 44	3,412 39	49,680 37
1877	6,721 75	38,408 15	3,776 22	48,906 12
1878	5,907 94	37,573 59	4,532 63	48,014 16
1879	5,181 21	38,638 09	2,239 54	46,058 84
1880	4,575 17	39,729 07	1,853 51	46,157 75
1881	5,841 92	44,470 44	2,217 46	52,529 82
1882	5,724 46	50,845 97	3,350 02	59,920 45
1883	5,833 44	53,889 77	3,342 63	63,075 84
1884	6,599 63	52,868 70	3,450 87	62,918 20
1885	7,735 99	53,295 96	3,302 55	64,334 50
1886	7,805 13	53,322 14	2,721 23	63,848 50
1887	8,748 17	54,003 91	3,365 92	66,118 00
1888	7,986 49	58,435 75	3,495 87	69,918 11
1889	10,102 79	56,567 12	2,541 08	69,210 99
1890	11,813 52	59,510 95	2,433 08	73,757 72
1891	13,932 46	66,900 99	4,619 75	85,822 66
1892	15,215 50	74,108 34	12,913 43	102,237 27
1893	15,440 11	78,406 53	75,798 88	169,645 52
1894	18,616 92	74,159 85	86,234 57	179,011 34
1895	21,818 43	84,902 31	34,649 47	141,370 21
1896	17,625 93	86,114 44	18,715 88	122,456 25
1897	16,703 72	90,888 11	31,061 67	138,653 50
1898	17,814 91	101,714 57	28,454 17	147,983 65
1899	16,593 80	100,375 11	95,260 99	212,229 90
1900	19,024 82	108,069 96	56,209 38	183,304 16
1901	15,729 09	100,348 78	5,222 46	121,300 33
1902	15,095 79	106,940 28	7,013 37	129,949 44
1903	18,940 31	115,452 93	7,743 88	142,137 12
1904	20,584 25	125,173 28	44,750 07	190,507 60
1905	20,970 06	127,193 35	37,826 75	185,990 34
1906	20,479 49	136,224 67	44,079 13	200,783 29
1907	21,857 04	143,655 53	78,164 88	243,677 45
1908	21,993 64	145,251 95	37,077 46	204,323 05
1909	16,604 27	147,740 68	17,863 93	182,208 88
1910	16,543 53	150,511 13	27,720 46	194,775 12
1911	20,971 33	166,941 77	57,847 95	245,761 05
1912	20,555 80	174,494 49	48,057 13	243,107 42

NATIVITY OF PATIENTS.

ADMITTED DURING 1912.	ADMITTED SINCE OPENING THE INSTITUTION.
United States.....	3,236
Ireland.....	207
Germany.....	218
England.....	194
Scotland.....	48
Sweden and Norway.....	90
Italy.....	74
Poland.....	314
Other countries.....	52
Total.....	4,433
United States.....	53,514
Ireland.....	17,292
Germany.....	9,352
England.....	8,613
Scotland.....	2,196
Sweden and Norway.....	3,456
Italy, from January 1, 1912.....	74
Poland, from January 1, 1912.....	314
Other countries.....	7,264
Total.....	102,075

E. F. LEIPER,
Superintendent.

REPORT OF CASES TREATED IN THE RECEIVING WARD AND REFERRED TO THE DISPENSARY, 1912.

No. of Cases.	No. of Cases.	No. of Cases.
Abrasions..... 131	Electric..... 21	Malar..... 1
Abscess:—	Fire..... 159	Mandible..... 1
Alveolar..... 9	Iodine..... 1	Metacarpal..... 3
Arm..... 9	Lime..... 4	Nasal..... 4
Axillary..... 3	Metal..... 1	Phalanx..... 2
Breast..... 1	Powder..... 36	Radius..... 1
Back..... 2	Scalds..... 37	Radius and ulna.. 6
Buttock..... 2	Sun..... 7	Tibia..... 1
Cervical..... 18	Bursitis..... 12	Toe..... 3
Chest..... 2	Calculi:—	Simple:—
Cornea..... 1	Vesical..... 1	Carpal..... 3
Ear..... 2	Callus..... 2	Clavicle..... 31
Eyebrow..... 1	Carbuncle..... 23	Coccyx..... 1
Face..... 5	Carious teeth..... 4	Colles'..... 87
Finger..... 15	Cellulitis..... 22	Femur..... 3
Foot..... 3	Chorea..... 2	Fibula..... 5
Head..... 1	Coccygodynia..... 1	Frontal bone..... 1
Ilium..... 1	Concussion..... 5	Malleolus, external 4
Inguinal..... 4	Conjunctivitis..... 16	Humerus:—
Ischio-rectal..... 3	Constipation..... 17	Anatomical neck 5
Jaw..... 4	Contractures from	External con-
Knee..... 2	burns..... 1	dyle..... 7
Leg..... 3	Contusions:—	Internal condyle 27
Palmar..... 9	Head and face..... 177	Shaft..... 10
Periauricular..... 2	Lower extremity... 250	Supracondylar.. 6
Peritonsillar..... 5	Trunk..... 148	Internal and ex-
Popliteal..... 1	Upper extremity... 420	ternal condyle,
Scalp..... 3	General..... 4	with fracture
Tubo-ovarian..... 1	Coxalgia..... 1	olecranon
Wrist..... 6	Cystitis..... 1	process..... 1
Adenitis:—	Deflected septum.... 2	Surgical neck... 12
Axillary..... 7	Deformity of foot.... 1	"T" Fracture.. 1
Femoral..... 3	Duodenal ulcer..... 1	Maxilla:—
Cervical..... 17	Delirium tremens, in-	Inferior..... 8
Inguinal..... 6	cipient..... 1	Superior..... 1
Adhesions..... 1	Edema of scrotum.... 1	Metacarpal..... 41
Alcoholism..... 70	Electric shock..... 1	Metatarsal..... 19
Amenorrhœa..... 2	Endometritis..... 1	Nose..... 14
Anæmia..... 1	Enlarged turbinate... 1	Olecranon..... 6
Angina pectoris..... 1	Enteritis..... 10	Phalanx..... 26
Aneurysm, traumatic.. 1	Epilepsy..... 18	Potts'..... 4
Ankylosis..... 1	Epiphyseal separation. 15	Radius..... 40
Appendicitis..... 7	Epistaxis..... 21	Ribs..... 49
Arterio-sclerosis..... 1	Erysipelas..... 3	Scapula:—
Arthritis:—	Exostosis..... 1	Accromion proc-
Acute..... 4	Fistula in ano..... 1	ess..... 3
Chronic..... 7	Foreign bodies:—	Body..... 1
Rheumatoid..... 3	Arm..... 3	Coracoid proc-
Traumatic..... 4	Ear..... 15	ess..... 2
Tuberculous..... 1	Esophagus..... 10	Skull..... 2
Avulsion of:—	Eye..... 339	Tibia..... 3
Finger nail..... 17	Finger..... 55	Ulna..... 13
Toe nail..... 5	Foot..... 16	Radius and ulna.. 41
Bites:—	Hand..... 38	Tibia and fibula.. 3
Cat..... 16	Intestines..... 1	Furuncle..... 93
Dog..... 154	Leg..... 9	Ganglion..... 3
Frost..... 8	Nose..... 8	Gastritis..... 45
Horse..... 10	Pharynx..... 6	Gastroenteritis... 6
Human..... 8	Side..... 1	Glaucoma..... 1
Insect..... 31	Spine..... 1	Heat exhaustion... 11
Bronchitis..... 11	Stomach..... 1	Hematuria..... 1
Bunions..... 3	Toe..... 1	Hematoma..... 13
Burns:—	Fractures:—	Hemophilia..... 2
Acid..... 24	Compound:—	Hemiplegia (old).... 1
Brush..... 16	Finger..... 3	Hemorrhage:—
		Secondary.....

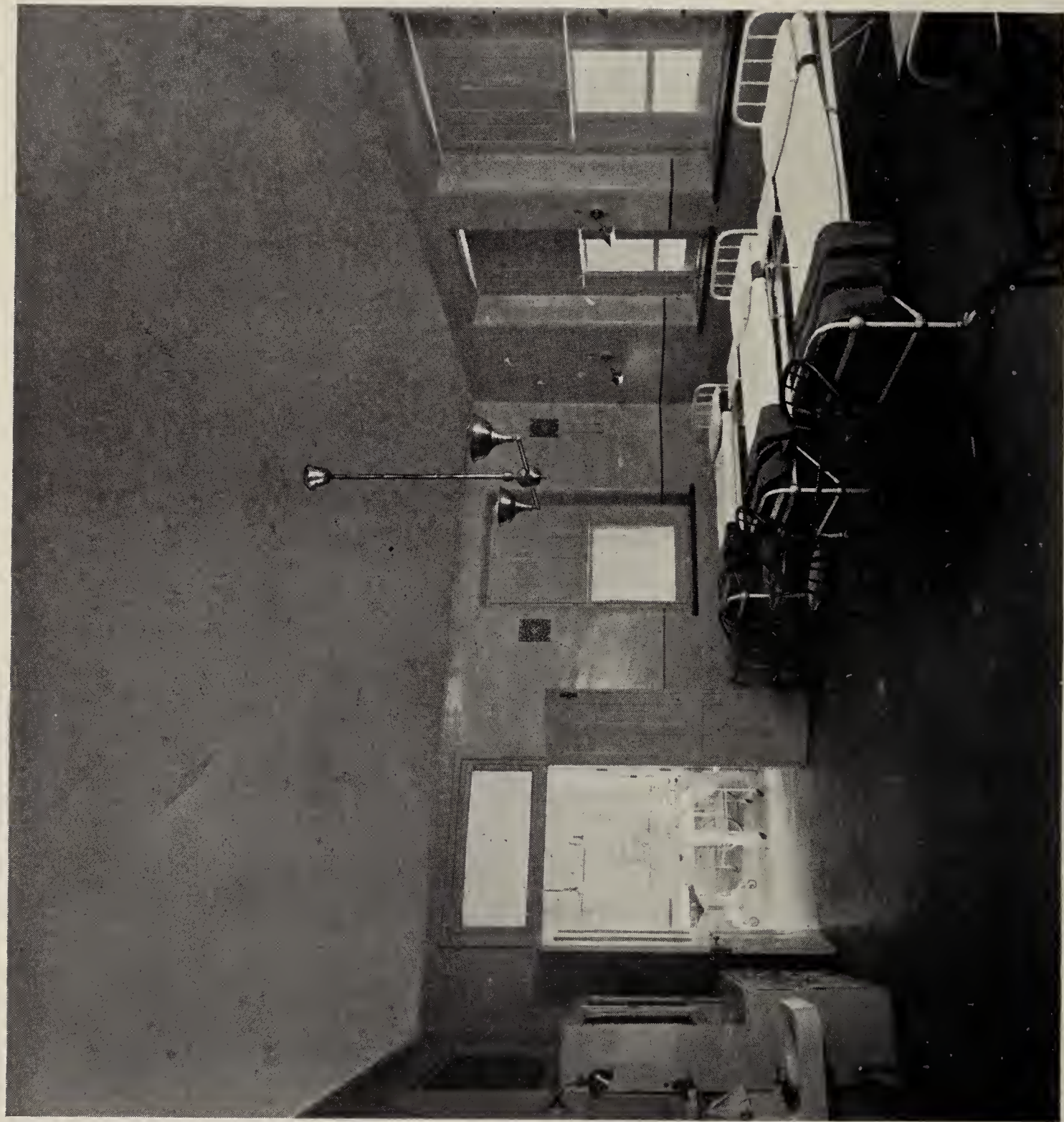


FIG. 15.—Receiving Ward, Women's Department

RECEIVING WARD—Continued.

No. of Cases.	No. of Cases.	No. of Cases.
Hemorrhoids:—	Opium..... 1	Uterus:—
External..... 2	Paris green..... 1	Retroversion..... 1
Internal..... 1	Rhus toxicodendron. 51	Vaccina..... 3
Hernia:—	Pregnancy..... 4	Vaccination..... 28
Inguinal..... 5	Prolapse of rectum... 4	Varicocele..... 1
Incisional..... 1	Prostatic enlargement. 3	Varicose ulcers..... 15
Hordeolum..... 6	Pyorrhœa alveolaris... 1	Varicose veins..... 7
Hydrocele..... 5	Retention of urine.... 5	Vertigo..... 5
Hysteria..... 16	Rheumatism:—	Venereal diseases:—
Impacted cerumen... 12	Acute..... 5	Chancroid..... 7
Indigestion..... 11	Chronic..... 9	Orchitis..... 4
Influenza..... 2	Rhinitis..... 1	Syphilis:—
Ingrowing toe nail... 8	Rupture:—	Primary..... 6
Insanity..... 2	Muscles..... 1	Secondary..... 20
Iritis..... 1	Vessels..... 2	Tertiary..... 1
Keratitis, ulcerative... 4	Salpingitis..... 2	Urethritis, Gono-
Laryngitis..... 8	Sciatica..... 2	coccic..... 12
Luxations:—	Singultus..... 3	Vaginitis..... 5
Ankle..... 1	Skin diseases:—	Verruca..... 2
Clavicle..... 8	Dermatitis..... 10	Wounds:—
Elbow..... 15	Eczema:—	Crushed:—
Finger..... 21	Rubrum..... 2	Finger..... 21
Mandible..... 3	Squamosum..... 15	Foot..... 2
Metacarpus..... 2	Erythema multi-	Thumb..... 4
Radius..... 3	forme..... 2	Toe..... 7
Sacro-iliac joint... 1	Herpes:—	Gunshot..... 11
Semilunar cartilage.. 5	Simplex..... 1	Incised:—
Shoulder..... 13	Zoster..... 4	Head and face.... 15
Thumb..... 11	Impetigo contagiosa. 10	Lower extremity.. 10
Ulna..... 1	Pediculosis:—	Upper extremity.. 33
Wrist..... 2	Capitis..... 4	Infected:—
Lymphangitis..... 3	Corporis..... 3	Head and face.... 30
Malingering..... 7	Pubis..... 3	Lower extremity.. 32
Malnutrition..... 1	Pruritus..... 1	Trunk..... 3
Mastitis..... 1	Scabies..... 17	Upper extremity.. 318
Mastoiditis..... 3	Sycosis vulgaris.... 1	Lacerated:—
Melancholia..... 1	Tinea versicolor.... 2	Head and face.... 857
Mitral regurgitation... 3	Urticaria..... 8	Lower extremity.. 168
Morton's disease..... 1	Verucca..... 3	Neck..... 1
Myocarditis..... 4	Sprains:—	Tongue..... 4
Myelitis..... 1	Lower extremity.... 145	Trunk..... 17
Necrosis:—	Trunk..... 25	Upper extremity.. 1012
Ribs..... 1	Upper extremity.... 127	Punctured:—
Nephritis, chronic.... 6	Synovitis..... 8	Head and face.... 9
Nephrolithiasis..... 1	Talipes equino-varus... 4	Lower extremity.. 55
Neuralgia..... 5	Tenosynovitis..... 16	Trunk..... 21
Neuritis..... 5	Thecitis..... 2	Upper extremity.. 41
Neurosis, occupation.. 1	Tongue-tie..... 1	Stab..... 3
Odontalgia..... 13	Tonsillitis..... 16	Traumatic amputa-
Ophthalmia neonato-	Torticollis..... 5	tions:—
rum..... 1	Tuberculosis, pulmon-	Fingers..... 71
Osteomyelitis..... 1	ary..... 7	Toe..... 1
Osteoperiostitis..... 2	Tumors:—	Wrist drop..... 1
Otitis media..... 8	Benign:—	Cases referred to:—
Paralysis:—	Fibroma..... 1	Ear, Nose and Throat
Musculo-spiral..... 9	Lipoma..... 4	Dispensary..... 8
Paraphimosis..... 2	Polyp..... 1	Eye Dispensary.... 12
Paronychia..... 15	Cysts:—	Medical Dispensary. 28
Periostitis..... 2	Chalazion..... 4	Teeth extraction... 2
Pertussis..... 2	Sebaceous..... 7	Diagnosis unclassified. 139
Pes planus..... 6	Malignant:—	Eloped..... 1
Pharyngitis..... 10	Carcinoma..... 2	Examination negative. 8
Phimosis..... 13	Epithelioma..... 3	Refused treatment.... 3
Pleurisy..... 13	Sarcoma..... 1	Total..... 7346
Pleurodynia..... 8	Ulcers..... 14	
Poisoning:—	Corneal..... 3	
Gas..... 1	Uræmia..... 2	
Iodine..... 1	Urinary incontinence.. 1	
Kerosene..... 2		

ABSTRACT OF HOUSE CASES.

MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Blood.</i>										
Anæmia:—										
Chlorosis.....	1	1	2	2	2
Pernicious.....		1	1	1	1
Secondary.....		4	2	2	1	1	2	4
Angio-neurotic œdema.....		1	1	1	1
Leukæmia:—										
Lymphatic, acute.....		2	2	1	1	2
Purpura:—										
Purpura simplex.....		1	1	1	1
Peliosis rheumatica.....		1	1	1	1
Septicæmia.....		1	1	1
<i>Bones and Joints.</i>										
Arthritis:—										
Acute multiple.....		1	1	1	1
of ankle.....		3	1	1	1
Gonococcic.....		2	1	2	1	3
with kerato-iritis.....	1	1	1	1	1
Chronic, of knee.....		1	1	1	1
Sacro-iliac sprain.....		1	1	1	1
Sprain of ankle.....		1	1	1	1
Charcot joint.....		2	1	1	1
Potts' disease.....		1	2	1	1	2
with pulmonary tuberculosis.....		1	1	1	1
Pes planus.....		1	1	1
Congenital dislocation of hip.....	1	1	1	1
Fracture of femur.....	1	1	1	1
<i>Circulatory System.</i>										
Arteries:—										
Aneurysm aorta.....	1	6	7	5	1	1	7
with suppurative pericarditis.....		1	1	1	1
Arterio-sclerosis.....	1	5	4	2	5	1	6
with cholelithiasis.....		1	1	1	1
with chronic nephritis.....		1	1	1	1
with chronic nephritis and myo- carditis.....		1	1	1	1
Heart:—										
Pericarditis:—										
Acute.....		1	1	1	1
Chronic.....		1	1	1	1
Endocarditis:—										
Acute.....		1	1	1	1
Malignant.....		1	1	1	1
with pericarditis.....		1	1	1	1
Chronic.....		3	3	3	3
with infarct of lung.....		1	1	1	1
with chronic nephritis.....	1	1	1	1
with myocarditis.....		1	1	1	1



FIG. 16.—Men's Medical Ward.



FIG. 17.—Women's Surgical Ward "A."

ABSTRACT OF HOUSE CASES—Continued.
MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Male.	Admitted, 1912.	Female,	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Circulatory System (Con.)</i>										
Valvular Diseases:—										
Aortic regurgitation.....	1	2	3	2	1	3
Mitral regurgitation.....	2	23	12	13	1	14	1	7	2	25
with chronic nephritis and hydrothorax.....	1	1	1	1
with chronic nephritis and myocarditis.....	1	1	1	1
with chronic interstitial nephritis...	1	1	1	1	2	2
with diffuse nephritis.....	7	4	3	4	3	7
with cirrhosis of liver.....	1	1	1	1
with myocarditis.....	2	1	1	2	2
Mitral stenosis.....	3	2	1	2	1	3
Combined Valvular Lesions:—										
Aortic disease, double.....	2	2	1	1	2
with hepatic cirrhosis.....	1	1	1	1
with mitral regurgitation.....	4	3	1	3	1	4
with mitral double.....	4	4	2	2	4
Aortic regurgitation and mitral regurgitation.....	3	2	1	2	1	3
and mitral stenosis.....	1	1	1	1
Aortic stenosis and mitral regurgitation.....	1	1	1	1
Mitral disease, double.....	9	5	4	6	3	9
with jaundice.....	1	1	1	1
with aortic regurgitation.....	1	1	1	1
with aortic stenosis.....	1	1	1	1
with adherent pericarditis.....	1	1	1	1
with chronic nephritis.....	1	1	1	1
Auricular fibrillation and hydrothorax..	1	1	1	1
Acute dilatation.....	2	1	1	2	2
Myocarditis.....	5	39	27	17	2	26	21	7	8	44
with angina pectoris.....	1	1	1	1
with cholelithiasis.....	1	1	1	1
with cirrhosis of liver.....	1	1	2	2	2
with hydrothorax.....	4	2	2	1	3	4
with chronic intersitital nephritis...	1	1	1	1	2	2
with chronic diffuse nephritis.....	3	3	2	1	3
with chronic diffuse nephritis and uremia.....	1	1	1	1
Veins:—										
Phlebitis and pulmonary infarct.....	1	1	1	1
<i>Digestive System.</i>										
Appendix:—										
Appendicitis, acute.....	1	1	1	1
with abscess.....	1	1	1	1
Chronic.....	1	1	1	1
Intestines:—										
Constipation.....	7	4	3	4	3	7
Obstruction and peritonitis.....	1	1	1	1
Post-operative adhesions.....	1	1	1	1
Enteritis, acute.....	1	11	5	7	6	1	5	12
and thrush.....	1	1	1	1
with varicocele and purpura.....	1	1	1	1
Colitis.....	2	2	2	2
Enterocolitis.....	7	4	3	3	1	2	1	7

ABSTRACT OF HOUSE CASES—Continued. MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Digestive System (Con.)</i>										
Parasites:—										
Taenia saginata.....		6	2	4	4	1	1			6
Whip-worms.....		1		1		1				1
Tumors:—										
Carcinoma of ascending colon.....		1		1			1			1
Carcinoma of rectum and liver.....		1	1					1		1
Liver:—										
Abscess.....		1	1				1			1
Cholangitis.....		3	2	1	3					3
and secondary anemia.....		1		1		1				1
Cholecystitis.....		4	3	1	2		1		1	4
Cholelithiasis.....		1	1			1				1
Cirrhosis, atrophic.....	1	8	6	3		4	2	2	1	9
hypertrophic.....		1		1		1				1
Hepatitis, syphilitic.....		1	1			1				1
Jaundice:—										
Catarrhal.....		1	1		1					1
Obstructive.....		1		1		1				1
Stomach and Duodenum:—										
Gastropotosis.....		3	1	2		2	1			3
Gastritis:—										
Acute.....		7	7		4	3				7
Chronic.....		5	3	2	3	1			1	5
Gastroenteritis.....		22	10	12	18	2		2		22
Ulcer, duodenal.....		3	2	1		2		1		2
Cancer:—										
Stomach.....		1	1				1			1
and liver.....		1	1					1		1
Cyclic vomiting.....		1	1		1					1
Salivary glands:—										
Mumps.....		1	1		1					1
Pharynx:—										
Pharyngitis.....		1		1		1				1
Pancreas:—										
Pancreatitis and jaundice.....		1	1					1		1
Tonsils:—										
Follicular tonsillitis.....		5		5	4	1				5
Peritonsillitis.....		1		1	1					1
Enlarged tonsils.....		2	2		1	1				2
<i>Genito-Urinary System.</i>										
Bladder:—										
Cystitis.....		2	1	1	1	1				2
and pelvic mass.....		1		1			1			1
Stone and pyelonephritis.....	1		1					1		1
Urethra:—										
Urethritis.....		1	1				1			1
Kidney:—										
Nephritis:—										
Chronic interstitial.....	1	12	4	9		7		5	1	13
with epistaxis.....		1		1					1	1
with myocarditis.....		2		2		1		1		2
with retino-choroiditis.....		1		1		1				1
with uræmia.....		2	2			1		1		2
with confusional insanity.....		1	1					1		1

ABSTRACT OF HOUSE CASES—Continued. MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Genito-Urinary System (Con.)</i>										
Kidneys:—										
Nephritis:—										
Chronic parenchymatous.....	10	7	3	1	5	1	2	1	10	
with mitral stenosis.....	1	1	1	1	1	1	1	1	1	1
with uræmia.....	1	3	3	1	3	1	1	1	4	1
Diffuse.....	1	1	1	1	1	1	1	1	1	1
with angioneurotic œdema.....	1	1	1	1	1	1	1	1	1	1
with enlarged prostate.....	1	5	3	2	3	2	2	2	5	1
Acute.....	1	1	1	1	1	1	1	1	1	1
with mitral regurgitation.....	1	12	7	6	1	1	11	1	13	1
Uræmia.....	1	1	1	1	1	1	1	1	1	1
Nephroptosis.....	1	1	1	1	1	1	1	1	1	1
Nephrolithiasis.....	1	1	1	1	1	1	1	1	1	1
Pyelitis.....	1	3	1	3	2	2	2	2	4	1
with cystitis.....	1	1	1	1	1	1	1	1	1	1
Uterus:—										
Pregnancy.....	1	1	1	1	1	1	1	1	1	1
with hyperemesis.....	2	2	2	2	2	2	2	2	2	2
with ascending paralysis.....	1	1	1	1	1	1	1	1	1	1
Abortion, inevitable.....	2	2	2	2	2	2	2	2	2	2
Fibroid.....	1	1	1	1	1	1	1	1	1	1
<i>Glandular System.</i>										
Thyroid:—										
Goitre:—										
Cystic.....	1	1	1	1	1	1	1	1	1	1
Parenchymatous.....	1	1	1	1	1	1	1	1	1	1
Graves' disease.....	3	3	3	3	2	2	1	1	3	3
Spleen:—										
Infarct.....	1	1	1	1	1	1	1	1	1	1
Lymph glands:—										
Adenitis, cervical.....	1	1	1	1	1	1	1	1	1	1
Hodgkin's disease.....	1	1	1	1	1	1	1	1	1	1
<i>Infections.</i>										
Diphtheria, nasal.....	1	1	1	1	1	1	1	1	1	1
Influenza.....	3	2	1	1	1	1	1	1	3	3
Malaria:—										
Aestivo-autumnal.....	2	15	16	1	14	2	1	1	17	2
Tertian.....	29	24	5	28	2	2	2	2	29	2
Double tertian.....	2	2	2	2	2	2	2	2	2	2
Measles.....	1	1	1	1	1	1	1	1	1	1
Parotitis.....	1	1	1	1	1	1	1	1	1	1
Rheumatism, acute articular.....	42	22	20	32	7	3	3	3	42	6
with acute endocarditis.....	6	4	2	2	3	3	3	3	6	1
with malignant endocarditis.....	1	1	1	1	1	1	1	1	1	1
with acute endocarditis and pericarditis.....	1	1	1	1	1	1	1	1	1	1
with chronic endocarditis and pericarditis.....	1	1	1	1	1	1	1	1	1	1
with acute pericarditis and pleural effusion.....	1	1	1	1	1	1	1	1	1	1
with orchitis.....	1	1	1	1	1	1	1	1	1	1
chronic.....	1	5	2	4	5	5	5	5	6	8
muscular.....	8	6	2	5	3	3	3	3	8	8

ABSTRACT OF HOUSE CASES—Continued. MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Infections (Con.)</i>										
Syphilis:—										
Congenital.....		1	1	1	1
Primary labial.....		1	...	1	...	1	1
Secondary.....		2	2	2	2
Tuberculosis, miliary.....		1	1	1	1
Typhoid:—										
Uncomplicated.....	3	84	63	24	71	2	2	12	87
Typhoid, complicated with:—										
Abscess, double parotid.....		1	...	1	1	1
popliteal.....		1	1	...	1	1
arm.....		1	1	...	1	1
Arthritis of shoulder.....		1	1	...	1	1
Bronchitis.....		1	1	1	1
Cholecystitis.....		2	2	...	2	2
Jaundice.....		1	1	...	1	1
Intestinal hemorrhage.....		4	3	1	3	1	...	4
and abscess of breast.....		1	...	1	1	1
and cystitis and cholecystitis.....		1	...	1	1	1
and pneumonia.....		1	...	1	1	1
and hematemesis.....		1	1	1	1
Intestinal perforation.....		4	3	1	4	4
Pharyngitis and laryngitis, ulcerative.....		1	1	1	...	1
Phlebitis.....		2	2	...	2	2
Periostitis of humerus.....		1	1	...	1	1
Pleurisy.....		2	2	...	2	2
Pulmonary tuberculosis.....		1	...	1	1	1
Pyelitis.....		1	1	...	1	1
Recrudescence.....		2	2	...	2	2
Relapse and otitis media.....		1	1	...	1	1
Otitis media.....		1	1	...	1	1
Varicella.....		1	...	1	1	1
Vaccinia.....		2	2	...	2	1
<i>Intoxications.</i>										
Heat exhaustion.....		6	5	1	5	1	6
Inorganic poisons:—										
Arsenic.....		1	1	...	1	1
Hydrochloric acid.....		2	2	...	1	1	...	2
Iodine.....		1	1	...	1	1
Lead.....		27	26	1	15	12	27
with pernicious anæmia.....		1	1	1	1
Mercuric chloride.....		2	2	...	1	1	2
with carbolic acid and turpentine..		1	...	1	1	...	1
Potassium cyanide.....		1	1	1	...	1
Organic poisons:—										
Autointoxication.....		6	5	1	6	6
Carbolic acid.....		7	4	3	4	3	...	3
Creolin.....		1	...	1	1	1
Illuminating Gas.....		10	5	5	8	1	1	10
Opium.....		4	1	3	3	1	4
Ptomain.....		3	2	1	3	3
Nicotine.....		1	1	1	1
Kerosene.....		1	1	...	1	1

ABSTRACT OF HOUSE CASES—Continued. MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Metabolic and Constitutional Diseases.</i>										
Diabetes.....		3	1	3			2	1		3
Diabetic coma.....		1	1					1		1
Rheumatoid arthritis.....	3	11	7	7	1	6			7	14
Rhachitis.....		3	1	2	1	1			1	1
and hydrocephalus.....		1	1					1		1
Gout.....		1		1	1					1
Malnutrition.....	1	4	2	3	3			2		5
Marasmus.....		6	5	1			1	4	1	6
Inanition.....		5	3	2		1		4		5
Malnutrition and varicella.....		1	1			1				1
and mastoiditis.....		1	1			1				1
<i>Nervous System.</i>										
Brain meninges:—										
Meningitis, pneumococcus.....		1		1				1		1
Tuberculous.....		1		1				1		1
with miliary tuberculosis.....		1		1				1		1
with tuberculous pneumonia.....		1		1				1		1
Septic from iritis.....		1	1					1		1
Syphilitic.....		2	2			1	1			2
Brain:—										
Apoplexy.....	1	17	10	8		7		11		18
with uræmia and pneumonia.....		2	1	1				2		2
Hemiplegia.....	5	9	6	8	8	3	1	4	6	14
Thrombosis.....	1	1	2			1		1		2
Syphilis.....	1		1					1		1
Palsy, cerebral.....		1		1			1			1
Insanity:—										
Acute mania.....		1	1				1			1
Delirium tremens.....		5	5		2	1		2		5
Dementia præcox.....		2	1	1			2			2
Paretic dementia.....		1	1				1			1
Melancholia.....		2	1	1		2				2
Tumor.....		1	1				1			1
Spinal cord:—										
Paraplegia.....		1	1					1		1
Poliomyelitis, anterior.....	2	3	4	1	1	3	1			5
Paralysis, acute ascending.....		1	1					1		1
Tabes dorsalis.....		3	1	2					3	3
General classification:—										
Alcoholism.....		20	17	3	13	5	2			20
Chorea.....	1	17	9	9	11	5	1	1		18
Insaniens.....		1		1					1	1
with endocarditis.....		1		1	1					1
with endocarditis and neuritis.....		1		1		1				1
Huntingdon's.....		1		1			1			1
Epilepsy.....		10	7	3	2	7			1	10
status epilepticus.....		1		1				1		1
and psychosis.....		1	1			1				1
Hysteria.....	1	7	1	7	2	5			1	8
Headache.....		1	1			1				1
Muscular dystrophy.....	1		1					1		1
Neurasthenia.....		13	2	11	4	5	2		2	13
post-operative.....		3	2	1		3				3
with nephroptosis.....		1		1		1				1

ABSTRACT OF HOUSE CASES—Continued. MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted 1912.	Male.	Female.	Recovered.	Improved.	Not improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Nervous System (Con.)</i>										
General classification (Con.):—										
Paralysis agitans.....	3	3	2	1	3
Senility.....	2	3	5	1	2	2	5
Vertigo.....	2	2	1	1	2
Peripheral nerves:—										
Neuralgia:—										
Intercostal.....	1	1	1	1
Trifacial.....	1	1	1	1
Neuritis:—										
Brachial.....	2	2	2	2
Multiple.....	1	2	1	2	1	1	1	3
Sciatic.....	1	10	7	4	4	4	3	11
Musculo-spiral.....	1	1	1	1
<i>Respiratory System.</i>										
Trachea and bronchi:—										
Asthma.....	9	7	2	2	7	9
Bronchitis:—										
Acute.....	3	31	16	18	26	5	1	2	34
Capillary.....	1	1	1	1
with gastritis.....	1	1	1	1
with varicella.....	2	1	1	1	1	2
Chronic.....	3	3	2	1	3
Bronchiectasis.....	1	2	3	1	2	3
Larynx:—										
Laryngitis.....	1	1	1	1
Spasmodic croup.....	1	1	1	1
Lungs:—										
Pneumonia:—										
Broncho.....	3	6	2	7	8	1	9
with asthma.....	1	1	1	1
Lobar.....	6	81	54	33	55	2	20	10	87
with pulmonary abscess.....	1	1	1	1
with delirium tremens.....	1	1	1	1
with delayed resolution.....	3	2	1	1	2	3
with empyema.....	3	2	1	2	1	3
with chronic endocarditis.....	1	1	1	1
with malignant endocarditis and premature labor.....	1	1	1	1
with pregnancy.....	1	1	1	1
with pleurisy.....	3	2	1	1	2	3
with pleural effusion and mitral stenosis.....	1	1	1	1
with otitis media.....	1	1	1	1	2	2
with typhoid fever.....	1	1	1	1
with nephritis.....	1	1	1	1
with hematoma of sheath of rectus.....	2	2	1	1	2

ABSTRACT OF HOUSE CASES—Continued. MEDICAL WARDS.

DISEASES.	Remaining Jan. 1, '12.	Admitted 1912.	Male.	Female.	Recovered.	Improved.	Not improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Respiratory System (Con.)</i>										
Lungs:—										
Pulmonary tuberculosis.....		35	21	14	15	6	4	10	35
with appendicitis.....		1	1	1	1
with apoplexy.....	1	1	1	1
with lobar pneumonia.....	1	1	1	1
with mitral stenosis.....		1	1	1	1
with pleural effusion.....		2	1	1	1	1	2
with Pott's disease.....		1	1	1	1
with pregnancy.....		1	1	1	1
with puerperium.....		1	1	1	1
Emphysema.....	1	1	1	1	2	2
<i>Serous Membranes and Cavities.</i>										
Peritoneum:—										
Peritonitis.....		2	2	2	2
Tuberculous.....		1	1	1	1
Pleura:—										
Pleurisy:—										
Acute.....	2	14	9	7	9	6	1	16
Chronic.....		1	1	1	1
Fibrinous.....		1	1	1	1
with effusion.....		13	9	4	5	5	2	1	13
Tuberculous.....		1	1	1	1
Empyema.....		6	6	1	3	1	1	6
Interlobar.....		1	1	1	1
Encysted.....		1	1	1
Pleurodynia.....		1	1	1	1
Multiple serositis.....	1	1	1	1
<i>Skin.</i>										
Dermatitis, exfoliative.....		2	1	1	1	1	2
Eczema:—										
Papulo-squamous.....		2	2	1	1	2
Hands.....		1	1	1	1
Vulva.....		1	1	1	1
Furunculosis.....		1	1	1	1
Psoriasis.....		1	1	1	1
<i>Soft Parts.</i>										
Abscess:—										
Arm.....		1	1	1	1
Ischio-rectal.....		1	1	1	1
Myositis.....		2	1	1	2	2
Muscle strain.....		1	1	1	1
Sarcomatosis.....		1	1	1	1
	81	1161	741	501	513	353	73	199	104	1242

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Medical Registrar.

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

I. SURGICAL DISEASES.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Alimentary System.</i>										
Appendix Vermiformis:—										
Inflammations:—										
Appendicitis:—										
Acute.....	7	59	39	27	54	6	1	5	66
with mesenteric and hepatic abscess.....	1	1	1	1
Gangrenous.....	7	7	7	7
with abscess.....	6	30	24	12	31	1	3	1	36
with peritonitis.....	29	18	11	21	1	7	29
Chronic.....	1	72	25	48	59	5	7	2	73
with cystic ovary.....	1	1	1	1
with Glenard's disease.....	1	1	1	1
with intestinal obstruction.....	1	1	1	1
Gall-bladder and biliary passages:—										
Cholelithiasis.....	1	25	7	19	14	6	1	3	2	26
Inflammations:—										
Cholecystitis.....	2	9	8	3	4	6	1	11
Pericholecystitis.....	4	1	3	2	1	1	4
Tumors:—										
Carcinoma of gall-bladder.....	1	1	1	1
Gums, alveolæ and teeth:—										
Inflammations:—										
Abscess, alveolar.....	4	4	3	1	4
Tumors:—										
Epulis.....	2	1	1	1	1	2
Intestine (including hernia):—										
Abnormalities and malformations:—										
Hernia:—										
Femoral.....	2	4	1	5	6	6
Inguinal:—										
Single.....	3	73	71	5	65	2	5	4	76
Double.....	12	11	1	10	2	12
Post-operative.....	12	7	5	9	2	1	12
Umbilical.....	1	2	1	2	2	1	3
Strangulated:—										
Femoral.....	4	1	3	3	1	4
Inguinal.....	10	7	3	7	2	1	10
Ventral through abdominal wall.....	1	1	1	1
Inflammations:—										
Typhoid fever.....	1	1	1	1
Typhoid perforation.....	3	3	3	3
Obstruction:—										
Acute.....	5	5	5	5
by gall-stones.....	1	1	1	1
Volvulus.....	1	1	1	1
Tumors:—										
Carcinoma ascending colon.....	2	1	1	2	2
Carcinoma descending colon.....	1	1	1	1
Carcinoma sigmoid.....	1	1	1	1
Lips:—										
Tumors:—										
Epithelioma.....	1	3	2	2	3	1	4

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Alimentary System (Con.)</i>										
Liver:—										
Inflammations:—										
Abscess.....		1	1	1	1
Cirrhosis.....		2	1	1	2	2
Tumors:—										
Carcinoma.....		2	1	1	2	2
Omentum:—										
Inflammations:—										
Abscess.....		1	1	1	1
Pancreas:—										
Inflammations:—										
Pancreatitis, chronic.....		3	3	1	1	1	3
Retrogressive and progressive tissue changes:—										
Diabetes mellitus.....		1	1	1	1
Tumors:—										
Carcinoma.....		3	3	1	2	3
Peritoneum:—										
Inflammations:—										
Abscess, retroperitoneal.....		1	1	1	1
Adhesions, post operative.....		9	2	7	4	3	1	1	9
Peritonitis:—										
Diffuse.....		1	1	1	1
Localized.....		1	1	1	1
Pelvic.....		3	3	1	1	1	3
Sinus, with post-operative insanity.....		1	1	1	1
Pharynx:—										
Inflammations:—										
Pharyngitis.....		1	1	1	1
Rectum and anus:—										
Abnormalities and malformations:—										
Hemorrhoids:—										
External.....		4	4	2	1	1	4
Internal.....		35	22	13	26	3	2	4	35
Combined.....		3	3	3	3
Prolapse.....		3	2	1	3	3
Inflammations:—										
Abscess, ischio-rectal.....	1	17	17	1	14	4	18
Fissure.....		3	3	3	3
Fistula.....		17	17	15	2	17
Tumors:—										
Carcinoma.....		4	1	3	1	1	1	1	4
Cyst, pilonidal.....		2	2	..	2	2
Epithelioma.....		1	1	1	1
Polyp.....		1	1	1	1
Stomach and duodenum:—										
Abnormalities and malformations:—										
Gastroptosis.....		2	2	1	1	2
Inflammations:—										
Adhesions, perigastric.....		1	1	1	1
Gastritis.....		10	7	3	3	6	1	10
Gastro-enteritis.....		1	1	1	1
Stenosis, pyloric.....		2	1	1	1	1	2

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Alimentary System (Con.)</i>										
Stomach and duodenum (Con.):—										
Inflammations (Con.):—										
Ulcers:—										
Gastric.....		7	5	2	3	2	1	1	7
with duodenal ulcer.....		1	1	1	1
Duodenal.....		3	3	3	3
perforated.....		3	3	1	1	1	3
Tumors:—										
Carcinoma of stomach.....	1	2	3	2	1	3
Lympho-sarcoma of stomach.....		1	1	1	1
Tongue and floor of mouth:—										
Inflammations:—										
Ludwig's angina.....		1	1	1	1
Tumors:—										
Carcinoma of floor of mouth.....		2	2	1	1	2
Epithelioma of tongue.....		3	1	2	1	2	1	3
Tonsils:—										
Abnormalities and malformations:—										
Hypertrophy.....		1	1	1	1
<i>Articulations.</i>										
Abnormalities and malformations:—										
Genu valgum.....		1	1	1	1
Hammer toe.....		2	2	2	2
Loose bodies in joint.....		1	1	1	1
Pes planus.....		2	1	1	2	2
Talipes equino-varus.....		2	1	1	1	1	2
Inflammations:—										
Gonococcic.....		5	4	1	4	1	5
Tuberculous:—										
Ankle.....		1	1	1	1
Hip.....	6	22	19	9	4	16	3	2	3	28
Knee.....	1	4	4	1	1	1	1	2	5
Wrist.....		1	1	1	1
Retrogressive and progressive tissue changes:—										
Arthritis, chronic hypertrophic....		3	2	1	3	3
<i>Blood-Vascular System.</i>										
Blood:—										
Abnormalities and malformations:—										
Hæmophilia.....		1	1	1	1
Inflammations:—										
Leukæmia, acute lymphatic.....		1	1	1	1
Septicæmia.....		1	1	1	1
Veins:—										
Abnormalities and malformations:—										
Varicose veins of leg.....	2	4	4	2	4	1	1	6
Tumors:—										
Hæmiangioma of foot.....		1	1	1	1

ABSTRACT OF HOUSE CASES—Continued. SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Generative Organs—Female.</i>										
Fallopian tubes:—										
Inflammations:—										
Hydrosalpinx.....	1	1	1	1
Double.....	...	1	...	1	1	...	1
Pyosalpinx, double.....	...	1	...	1	1	1
Double with residual abscess....	...	1	...	1	1	...	1
Ruptured.....	...	1	...	1	1	1
Salpingitis.....	7	44	...	51	36	7	7	...	1	51
with diffuse peritonitis.....	...	1	...	1	1	...	1
Salpingo-oophoritis.....	...	1	...	1	1	1
Ovaries:—										
Abnormalities and malformations:—										
Prolapse.....	...	1	...	1	1	1
Inflammations:—										
Abscess, tubo-ovarian.....	2	20	...	22	16	2	1	...	3	22
with intestinal obstruction.....	...	1	...	1	1	1
Bilateral.....	...	2	...	2	2	2
Tumors:—										
Cyst:—										
Calcified.....	...	1	...	1	1	1
Intraligamentous.....	...	1	...	1	1	1
Unilocular.....	1	1	...	2	2	2
Perineum:—										
Traumatism:—										
Lacerated perineum and cervix....	1	21	...	22	17	3	2	22
Lacerated perineum.....	...	9	...	9	7	2	9
Uterus:—										
Abnormalities and malformations:—										
Anteflexion.....	...	3	...	3	1	1	1	3
Didelphys.....	...	1	...	1	1	1
Prolapse.....	...	5	...	5	3	1	1	5
Retroflexion.....	...	5	...	5	4	...	1	5
Retroversion.....	...	15	...	15	11	4	15
Stenosis of cervix.....	...	4	...	4	2	1	1	4
Inflammations:—										
Cellulitis, pelvic.....	...	1	...	1	1	1
Endometritis.....	2	20	...	22	14	3	2	...	3	22
Septic, with pelvic abscess and sacculated empyema.....	...	1	...	1	1	...	1
Menorrhagia.....	...	1	...	1	1	...	1
Metrorrhagia.....	...	2	...	2	1	...	1	2
Tumors:—										
Carcinoma of cervix.....	...	2	...	2	...	1	1	2
of uterus.....	1	6	...	7	3	1	2	...	1	7
Epithelioma of cervix.....	...	1	...	1	1	1
Fibromyoma.....	...	15	...	15	8	1	4	2	...	15
Polyp.....	...	2	...	2	2	2
Sarcoma.....	...	1	...	1	1	1
Vagina:—										
Inflammations:—										
Abscess Bartholin's gland.....	...	2	...	2	2	2
Fistula, recto-vaginal.....	...	1	...	1	...	1	1
Vaginitis, gonococcic.....	...	4	...	4	...	4	4
Tumors:—										
Carcinoma anterior vaginal wall...	...	2	...	2	...	2	2

ABSTRACT OF HOUSE CASES—Continued. SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Generative Organs—Female (Con.)</i>										
Vulva:—										
Inflammations:—										
Abscess.....		1		1			1			1
Venereal warts.....		1		1				1		1
Traumatism:—										
Hæmatoma.....		1		1	1					1
Tumors:—										
Epithelioma.....		2		2		1		1		2
Pregnancy and complications:—										
Abortion, incomplete.....	2	56		58	55	2			1	58
Threatened.....		5		5	4	1				5
Eclampsia.....		14		14	5			9		14
Hemorrhage, post-partum.....		1		1	1					1
Infants:—										
New born.....		51	15	36	38	2		8	3	51
Still born.....		16	8	8				16		16
Labor.....	1	35		36	33	2	1			36
Obstructed.....		8		8	7				1	8
Nursing mother.....		1		1	1					1
Placenta prævia.....		2		2	2					2
Pregnancy.....		20		20	4	8	5		3	20
Extra-uterine.....		2		2	2					2
Ruptured.....		7		7	7					7
Cornual.....		1		1	1					1
Puerperal sepsis.....	1	15		16	3	1	1	9	2	16
Puerperium.....		1		1		1				1
Retained secundines.....		16		16	13	2	1			16
Toxæmia of pregnancy.....		1		1			1			1
Vomiting, pernicious.....		5		5	4	1				5
<i>Generative Organs—Male.</i>										
Epididymis:—										
Inflammations:—										
Epididymitis.....		2	2			2				2
Gonococcic.....		1	1						1	1
Penis and prepuce:—										
Abnormalities and malformations:—										
Hypospadias.....		1	1		1					1
Paraphimosis.....		3	3		3					3
Phimosis.....		50	50		47	2		1		50
Redundant prepuce.....		6	6		6					6
Inflammations:—										
Chancroids.....		3	3		2	1				3
Venereal warts.....		1	1			1				1
Prostate gland:—										
Inflammations:—										
Pyogenic.....		1	1			1				1
Tuberculous.....		2	2			1	1			2
Tumors:—										
Hypertrophy.....		13	13		2	2	5	4		13
Spermatic cord:—										
Abnormalities and malformations:—										
Varicocele.....	1	10	11		11					11

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Generative Organs—Male (Con.)</i>										
Testicle:—										
Abnormalities and malformations:—										
Undescended, with congenital hernia.....		1	1	1	1
Inflammation:—										
Orchitis, gonococcic.....		2	2	2	2
Tuberculous.....	1	1	1	1
Tunica vaginalis:—										
Inflammations:—										
Hydrocele.....		24	24	22	2	24
Encysted.....		1	1	1	1
Double.....		1	1	1	1
<i>Glandular System.</i>										
Mammary gland:—										
Inflammations:—										
Abscess.....		4	4	2	2	4
Mastitis, chronic.....		1	1	1	1
Tumors:—										
Carcinoma:—										
Adeno:—		1	1	1	1
Medullary.....		1	1	1	1
Recurrent.....	2	1	3	1	2	3
with lymphœdema of arm.....		1	1	1	1
Scirrhus.....		8	8	5	3	8
Cyst.....		1	1	1	1
Fibro-adenoma.....		2	2	2	2
Thyroid gland:—										
Tumors:—										
Goitre:—										
Exophthalmic.....		1	1	1	1
Parenchymatous.....		2	2	2	2
<i>Lymphatic System.</i>										
Lymph nodes:—										
Inflammations:—										
Adenitis, suppurative:—										
Axillary.....		2	2	1	1	2
Cervical.....	1	12	9	4	10	2	1	13
Inguinal.....		7	5	2	4	2	1	7
Submaxillary.....		5	5	2	1	1	1	5
Adenitis:—										
Axillary.....		1	1	1	1
Cervical.....	1	17	6	12	10	6	1	1	18
Femoral.....		2	2	1	1	2
Inguinal.....	1	8	9	6	3	9
Tumors:—										
Carcinoma axillary nodes.....		1	1	1	1
Inguinal nodes.....		1	1	1	1
Sarcoma cervical nodes.....		1	1	1	1
Inguinal nodes.....		1	1	1	1
Retroperitoneal.....		1	1	1	1

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Lymphatic System (Con.)</i>										
Lymph vessels:—										
Inflammations:—										
Elephantiasis.....		1	1	1	1
Lymphœdema of right leg.....		1	1	1	1
<i>Nervous System.</i>										
Brain:—										
Apoplexy.....		1	1	1	1
Bell's palsy with herpes zoster.....		1	1	1	1
Hemiplegia, spastic.....		1	1	1	1
Meningo-encephalitis.....		1	1	1	1
Brain; membranes:—										
Hydrocephalus, external.....		1	1	1	1
Meningitis:—										
Lepto, chronic.....		1	1	1	1
Septic.....		1	1	1	1
Syphilitic.....		3	2	1	2	1	3
Cerebro-spinal.....		1	1	1	1
Syphilis, cerebral.....		1	1	1	1
Cord, spinal:—										
Paralysis, acute ascending.....		1	1	1	1
Tabes dorsalis.....		2	1	1	1	1	2
General classification:—										
Epilepsy.....		1	1	1	1
Neurasthenia.....		3	3	2	1	3
Nerves, peripheral:—										
Neuritis.....		1	1	1	1
<i>Osseous and Cartilaginous System.</i>										
Abnormalities and malformations:—										
Coccygodynia.....		2	2	2	2
Polydactylism.....		1	1	1	1
Scoliosis.....		2	1	1	2	2
Inflammations:—										
Pyogenic:—										
Epiphysitis.....	1	1	2	2	2
Necrosis:—										
Femur.....		6	6	4	2	6
Manubrium.....		1	1	1	1
Maxilla.....		3	3	2	1	3
Metacarpal bones.....		2	1	1	1	1	2
Metatarsal bones.....		3	3	2	1	3
Rib.....		2	2	1	1	2
Skull.....		1	1	1	1
Tibia.....		8	8	1	7	8
Osteitis.....		1	1	1	1
Osteomyelitis:—										
Femur.....		3	3	2	1	3
Fibula.....		1	1	1	1
Tibia.....	1	2	3	1	1	1	3

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Osseous and Cartilaginous System (Con.)</i>										
Inflammations (Con.):—										
Pyogenic (Con.):—										
Periostitis:—										
Rib.....		1	...	1	1	1
Tibia.....		6	4	2	5	1	6
Tuberculous:—										
Tarsus.....		4	3	1	1	2	1	4
Vertebræ.....	1	16	10	7	...	6	6	1	4	17
Tumors:—										
Carcinoma of maxilla (recurrent)....		3	3	2	1	3
Cyst of humerus.....		1	1	...	1	1
Cyst of tibia.....		1	...	1	1	1
Exostosis.....		3	2	1	1	1	1	3
Osteo-sarcoma of maxilla.....		1	1	1	1
Osteo-sarcoma of occipital bones....	1	1	1	...	1
<i>Respiratory System.</i>										
Larynx:—										
Inflammations:—										
Laryngitis, acute.....		1	...	1	...	1	1
Tumors:—										
Fibroma of vocal cords.....		1	1	1	1
Lungs:—										
Inflammations:—										
Abscess.....		1	1	1	...	1
Pleura:—										
Inflammations:—										
Empyema.....	1	18	12	7	7	1	1	9	1	19
Pleurisy with effusion.....		1	1	1	1
Sinuses:—										
Inflammations:—										
Sinusitis, frontal.....		1	1	...	1	1
<i>Soft Parts.</i>										
Bursæ:—										
Inflammations:—										
Bursitis:—										
Post-olecranon.....	1	...	1	1	1
Prepatellar.....		5	3	2	2	3	5
Muscles:—										
Contractures:—										
Torticollis.....		2	2	...	2	2
Inflammations:—										
Lumbago.....		2	2	2	2
Skin and its appendages:—										
Abnormalities and malformations:—										
Ingrowing toenail.....		1	1	...	1	1
Infestation:—										
Pediculosis corporis.....		1	1	...	1	1
Scabies.....		1	1	...	1	1

ABSTRACT OF HOUSE CASES—Continued. SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Soft Parts (Con.)</i>										
Skin and its appendages (Con.):—										
Inflammations:—										
Dermatitis, iodoform.....		1	1	1	1
Eczema.....		4	1	3	4	4
Furunculosis.....	1	3	3	1	3	1	4
Syphilis.....	3	45	35	13	1	33	12	1	1	48
Tumors:—										
Carcinoma of face.....		2	1	1	1	1	2
Clavus.....		2	2	2	2
Cyst:—										
Branchial.....		1	1	1	1
Sebaceous.....		1	1	1	1
Enchondroma of ear.....		1	1	1	1
Epithelioma:—										
Eyelid.....		1	1	1	1
Face.....		1	1	1	1
Forehead.....		1	1	1	1
Nose.....	1	1	2	2	2
Fibroma:—										
Buttocks.....		1	1	1	1
Lipoma:—										
Back.....		1	1	1	1
Buttocks.....		1	1	1	1
Shoulder.....		2	2	2	2
Soft parts (in entirety):—										
Inflammations:—										
Abscess:—										
Abdominal wall.....		1	1	1	1
Arm.....		1	1	1	1
Back.....		2	2	1	1	2
Leg.....		2	2	1	1	2
Palmar.....		4	4	1	2	1	4
Popliteal.....		2	2	2	2
Thigh.....		1	1	1	1
Carbuncle.....	2	12	14	8	4	2	14
Cellulitis:—										
Arm.....		5	5	5	5
Eyelid.....		1	1	1	1
Face.....		2	1	1	2	2
Foot.....		3	2	1	3	3
Leg.....	1	8	9	3	5	1	9
Neck.....		2	2	1	1	2
Scalp.....		1	1	1	1
Gangrene.....		6	5	1	2	1	2	1	6
Senile.....		3	1	2	3	3
Painful stump.....		3	3	3
Ulcers:—										
Leg.....	2	5	2	5	3	3	1	7
Tumors:—										
Sarcoma:—										
Scalp.....		1	1	1	1
Thigh.....		2	2	1	1	2
Toe.....		1	1	1	1
Tendons:—										
Tumors:—										
Ganglion.....		3	3	2	1	3

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Urinary System.</i>										
Bladder:—										
Calculus.....	1	1	1	1
Inflammations:—										
Cystitis.....	1	5	5	1	4	2	6
tuberculous.....	3	2	1	3	3
Kidney:—										
Abnormalities and malformations:—										
Movable.....	1	1	1	1	1	1	2
Inflammations:—										
Nephralgia.....	1	1	1	1
Nephritis.....	1	1	1	1
Perinephric abcess.....	2	1	1	1	1	2
Pyonephrosis.....	3	3	1	1	1	3
Nephrolithiasis.....	1	11	8	4	6	3	3	12
Retrogressive and progressive tissue changes:—										
Cystic degeneration.....	1	1	1	1
Ureter:—										
Calculus.....	4	2	2	2	1	1	4
Urethra:—										
Abnormalities and malformations:—										
Fistula, urinary.....	1	1	1	1
Inflammations:—										
Abscess, periurethral.....	3	3	1	1	1	3
Urethritis.....	4	4	8	2	5	1	8
Stricture.....	1	9	10	4	5	1	10
Total.....	86	1582	847	821	1004	321	124	138	81	1668

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Surgical Registrar.

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

II. SURGICAL INJURIES.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Alimentary System.</i>										
Intestines:—										
Foreign body in.....		1	1	1	1
Rupture.....		3	2	1	1	2	3
Liver:—										
Stab wound of.....		1	1	1	1
Esophagus:—										
Acid burn.....		1	1	1	1
Foreign body in.....		3	1	2	3	3
Tongue and floor of mouth:—										
Lacerated wound of tongue.....		1	1	1	1
<i>Articulations.</i>										
Dislocations:—										
Semilunar cartilage of knee.....		3	3	3	3
Relaxation:—										
Sacro-iliac joint.....		1	1	1	1
Sprains:—										
Ankle.....		12	6	6	10	2	12
Back.....		2	1	1	1	1	1	2
Knee.....		1	1	1	1
Sacro-iliac joint.....		1	1	1	1
Traumatic arthritis:—										
Knee.....		2	1	1	2	2
Traumatic synovitis:—										
Knee.....		4	4	3	1	4
<i>Generative Organs—Male.</i>										
Orchitis:—										
Traumatic.....		2	2	1	1	2
<i>Glandular System.</i>										
Spleen:—										
Rupture of.....		1	1	1	1
<i>Nervous System.</i>										
Brain:—										
Concussion.....		31	24	7	23	7	1	31
General classification:—										
Epilepsy, traumatic.....		1	1	1	1

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Osseous and Cartilaginous System.</i>										
Fractures:—										
Head and trunk:—										
Skull:—										
Vault:—										
Compound comminuted.....		1	1	1	1
Depressed.....		6	6	4	1	1	6
Linear.....		1	1	1	1
Simple.....		5	5	4	1	5
Base.....		13	12	1	3	1	9	13
Bones of face:—										
Inferior maxilla:—										
Simple.....	1	8	8	1	1	7	1	9
Compound.....		1	1	1	1
Nasal bones:—										
Simple.....		2	1	1	2	2
Compound.....		1	1	1	1
Vertebrae:—										
Cervical.....		1	1	1	1
Dorsal.....	1	1	1	1
Lumbar.....		1	1	1	1
Ribs.....		10	10	4	3	3	10
Pelvic bones:—										
Pelvis.....		4	4	2	1	1	4
Upper extremity:—										
Scapula:—										
Accromion process.....		1	1	1	1
Humerus:—										
Shaft:—										
Simple.....		4	1	3	2	1	1	4
Compound.....		3	2	1	3	3
Supracondylar.....		1	1	1	1
Internal epicondyle.....		1	1	1	1
Lower extremity.....		6	3	3	2	2	2	6
Upper extremity.....		5	2	3	2	2	1	5
Radius:—										
Colles':—										
Simple.....		5	4	1	1	3	1	5
Shaft:—										
Simple.....	1	2	2	1	1	1	1	3
Both bones of forearm:—										
Simple.....		3	2	1	2	1	3
Compound.....		3	1	2	1	2	3
Metacarpals:—										
Simple.....		2	2	2	2
Compound.....		2	2	1	1	2
Lower extremity:—										
Femur:—										
Neck.....	3	22	14	11	11	5	1	3	5	25
Shaft:—										
Simple.....	2	23	20	5	17	1	2	5	25
Compound.....		2	2	1	1	2
Supracondylar.....		1	1	1	1

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Osseous and Cartilaginous System (Con.)</i>										
Fractures (Con.):—										
Lower extremity (Con.):—										
Fibula:—										
Shaft:—										
Simple.....	1	12	12	1	11	2	13
Compound.....	1	1	1	1
Patella:—										
Simple.....	12	8	4	9	2	1	12
Both patellæ.....	1	1	1	1
Tibia:—										
Shaft:—										
Simple.....	1	19	17	3	10	8	2	20
Compound.....	1	3	4	1	1	2	4
Tuberosity.....	1	1	1	1
Epiphyseal separation, lower end..	1	1	1	1
Tibia and fibula:—										
Shaft:—										
Simple.....	4	14	15	3	14	2	1	1	18
Compound.....	1	4	4	1	1	1	3	5
Multiple.....	1	1	1	1
Lower extremities:—										
Pott's fracture.....	2	22	20	4	11	4	2	7	24
Malleoli:—										
External.....	11	9	2	8	3	11
Internal.....	3	3	3	3
Both.....	1	2	3	2	1	3
Tarsus:—										
Astragalus.....	1	1	1	1
Calcaneum.....	4	3	1	2	2	4
Metatarsus:—										
Simple.....	2	2	2	2
Compound.....	3	3	2	1	3
Dislocations:—										
Ankle, compound.....	1	1	1	1
Clavicle, outer end.....	1	1	1	1
Elbow, posterior.....	3	2	1	1	1	1	3
Finger.....	3	3	2	1	3
Hip.....	5	4	1	3	1	1	5
Humerus, fracture dislocation, upper end.....	1	1	1	1
Shoulder.....	5	5	4	1	5
Thumb.....	4	4	3	1	4
<i>Soft Parts.</i>										
Burns and scalds:—										
Acid.....	1	1	1	1
Fire:—										
Local.....	17	6	11	8	6	1	2	17
General.....	21	11	10	21	21
Scalds.....	9	7	2	4	2	3	9

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Soft Parts (Con.)</i>										
Contusions:—										
Abdomen.....		6	5	1	4	1	1	6
Arm.....		1	1	1	1
Back.....		14	14	8	6	14
Chest.....		3	3	3	3
Face.....		2	2	2	2
Foot.....		8	8	7	1	8
Head.....		1	1	1	1
Hip.....	1	4	4	1	2	3	5
Knee.....		5	4	1	2	3	5
Leg.....		7	5	2	2	4	1	7
Perineum.....		1	1	1	1
Shoulder.....	1	1	1	1
Side.....		2	2	2	2
Thigh.....	1	1	2	1	1	2
Toe.....		1	1	1	1
General.....		21	14	7	14	7	21
Crush:—										
Arm.....	1	1	1	1
Fingers.....		1	1	1	1
Foot.....		1	1	1	1
Hand.....		1	1	1	1
Knee.....	1	1	1	1
Leg.....		3	3	1	2	3
Toe.....		1	1	1	1
Foreign bodies in:—										
Finger.....		1	1	1	1
Foot.....		3	1	2	3	3
Hand.....		3	3	1	2	3
Hæmatomata:—										
Abdomen.....		1	1	1	1
Occiput.....		2	2	2	2
Perineum.....		1	1	1	1
Thigh.....		1	1	1	1
Wounds:—										
Gunshot:—										
Abdomen.....		1	1	1	1
Chest.....		1	1	1	1
Foot.....		2	1	1	2	2
Head.....		5	5	2	2	1	5
Heart.....		1	1	1	1
Leg.....		3	3	1	1	1	3
Mouth.....		1	1	1	1
Neck.....		1	1	1	1
Shoulder.....		1	1	1	1
Vertebra.....		1	1	1	1
Thigh.....		2	2	2	2
Wrist.....		1	1	1	1
Incised:—										
Hand, with pneumonia.....		1	1	1	1
Throat.....		2	2	1	1	2

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Soft Parts (Con.)</i>										
Wounds (Con.):—										
Infected:—										
Arm.....		1	1	1	1
Face.....		1	1	1	1
Finger.....		7	6	1	4	3	7
Foot.....		7	7	4	2	1	7
Hand.....		5	5	2	1	1	1	5
Knee.....		5	5	2	1	2	5
Leg.....		7	7	5	1	1	7
Scalp.....		4	1	3	2	2	4
Thigh.....		1	1	1	1
with tetanus.....		2	2	1	1	2
Lacerated:—										
Arm.....		7	2	5	4	2	1	7
Face.....		2	2	1	1	2
Finger.....		1	1	1	1
Forearm.....		1	1	1	1
Foot.....		1	1	1	1
Hand.....		5	5	2	3	5
Leg.....		3	3	1	1	1	3
Lip.....		4	3	1	1	3	4
Scalp.....		23	18	5	16	7	23
Thigh.....		1	1	1	1
Punctured:—										
Chest.....		1	1	1	1
Foot.....		1	1	1	1
Neck.....		1	1	1	1
Stab:—										
Back.....		1	1	1	1
Leg and arm.....		1	1	1	1
Tendons:—										
Rupture:—										
Tendo-Achillis'.....		1	1	1	1
Severed:—										
Flexor, of fingers.....		3	3	2	1	3
<i>Urinary System.</i>										
Bladder:—										
Foreign body in.....		1	1	1	1
Kidney:—										
Contusion.....		2	2	1	1	2
Rupture.....		1	1	1	1
Urethra:—										
Foreign body in.....		1	1	1	1
Rupture.....		1	1	1	1
Total.....	26	619	505	140	342	166	21	66	50	645

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FIG. 18.—Lower Operating Room in the Ingersoll Building.



FIG. 19.—Upper Operating Room, Ingersoll Building.



FIG. 20.—Upper Sterilizing Room.

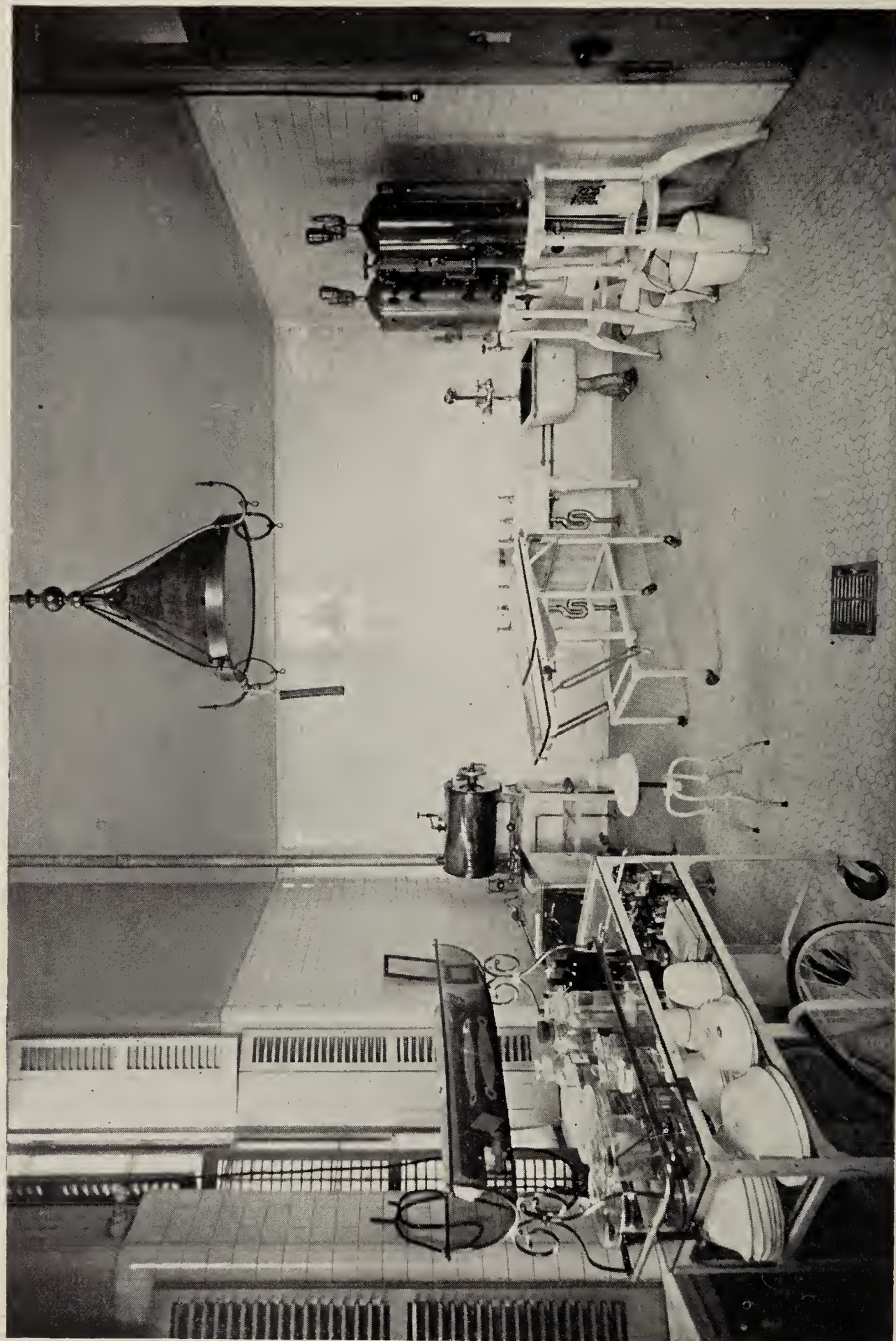


FIG. 21.—One of the Dressing Rooms for the Surgical Wards.

ABSTRACT OF HOUSE CASES—Continued,

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Diseases of the Eye.</i>										
Lids:—										
Abnormalities and malformations:—										
Ectropion.....		1	1	1	1
Entropion.....		1	1	1	1
Traumatism:—										
Laceration.....		1	1	1	1
Tumors:—										
Chalazion.....		2	2	2	2
Lachrymal apparatus:—										
Inflammations:—										
Abscess, double.....		1	1	1	1
Dacryo-cystitis.....	1	1	1	1	2	2
Fistula.....		1	1	1	1
Conjunctiva:—										
Abnormalities and malformations:—										
Pterygium.....		1	1	1	1
Inflammations:—										
Conjunctivitis.....		5	2	3	3	2	5
Ophthalmia neonatorum.....		1	1	1	1
Traumatism:—										
Burns:—										
Acid.....		4	4	1	3	4
Metal.....		3	3	1	2	3
Powder.....		1	1	1	1
Lacerations.....		1	1	1	1
Cornea and sclera:—										
Abnormalities and malformations:—										
Buphthalmus.....		1	1	1	1
Inflammations:—										
Hypopyon.....		1	1	1	1
Keratitis:—										
Bulbous.....	1	1	1	1
Interstitial.....		16	6	10	10	3	3	16
Nebular.....		1	1	1	1
Phlyctenular.....	1	4	3	2	1	4	5
Syphilitic.....		1	1	1	1
Traumatic.....		1	1	1	1
Tuberculous.....		1	1	1	1
Ulcerative.....	2	37	21	18	20	15	2	2	39
Vascular.....		1	1	1	1
Sclero-kerato-iritis.....	1	1	2	1	1	2
Ulcers.....		12	8	4	6	5	1	12
Traumatism:—										
Burns:—										
Acid.....		1	1	1	1
Lacerations:—										
Cornea and sclera.....		1	1	1	1
Sclera.....		1	1	1	1
Penetrating Wounds:—										
Cornea.....		1	1	1	1
Sclera.....		1	1	1	1

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Diseases of the Eye (Con.)</i>										
Eyeball and Orbit:—										
Inflammations:—										
Glaucoma:—										
Acute.....		6	6	4	1	1	6
Secondary.....	1	2	2	1	1	2	3
Panophthalmitis.....		4	1	3	1	3	4
Phthisis bulbi.....		4	2	2	1	2	1	4
Traumatism:—										
Contusion of eyeball.....	1	10	9	2	7	4	11
Foreign body in orbit.....		5	5	3	2	5
Gunshot wound of orbit.....		1	1	1	1
Laceration of eyeball.....		1	1	1	1
Penetrating wound of eyeball.....		1	1	1	1
Rupture of eyeball.....		1	1	1	1
Tumors:—										
Cystoid cicatrix.....		1	1	1	1
Iris and Ciliary Body:—										
Abnormalities and malformations:—										
Pupillary membrane.....		1	1	1	1
Inflammations:—										
Irido-cyclitis.....		3	3	1	2	3
Irido-dialysis.....		1	1	1	1
Iritis:—										
Acute.....	1	6	5	2	5	1	1	7
Chronic.....		2	2	1	1	2
Syphilitic.....		1	1	1	1
Traumatic.....		1	1	1	1
Kerato-iritis.....		6	3	3	1	5	6
Retina and Choroid:—										
Inflammations:—										
Atrophy, optic.....		6	6	2	4	6
Choroiditis.....		3	3	3	3
Embolism retinal artery.....		1	1	1	1
Neuritis.....		1	1	1	1
Neuro-retinitis.....		2	2	2	2
Retinitis:—										
Hemorrhagic.....	1	3	1	3	3	1	4
Retino-Choroiditis.....		1	1	1	1
Uveitis.....		2	2	1	1	2
Tumors:—										
Glioma of retina.....		1	1	1	1
Lens:—										
Cataract:—										
Capsular.....	1	3	2	2	1	3	4
Congenital.....		6	4	2	3	3	6
Secondary.....		12	6	6	8	4	12
Senile.....	6	70	39	37	31	39	1	1	4	76
Traumatic.....	1	10	10	1	3	8	11
Traumatism:—										
Dislocated lens.....	1	1	1	1	2	2
Muscles:—										
Strabismus:—										
Convergent.....		9	3	6	6	3	9
Divergent.....		4	3	1	3	1	4
	19	298	182	135	143	144	15	2	13	317

ABSTRACT OF HOUSE CASES—Continued.

SURGICAL WARDS.

DIAGNOSIS.	Remaining Jan. 1, '12.	Admitted, 1912.	Male.	Female.	Recovered.	Improved.	Not Improved.	Died.	Remaining Jan. 1, '13.	Total.
<i>Diseases of Ear, Nose and Throat.</i>										
Ear:—										
External ear:—										
Inflammations:—										
Furuncle of meatus.....		2	2	2	2
Middle Ear:—										
Inflammations:—										
Mastoid abscess.....		2	2	1	1	2
Mastoiditis, acute.....		22	14	8	14	6	1	1	22
with thrombosis lateral sinus..		2	2	1	1	2
Otitis media.....		5	3	2	4	1	5
with meningitis.....		1	1	1	1
Nose and Throat:—										
Empyema of antrum of Highmore....		1	1	1	1
Epistaxis.....		2	1	1	2	2
Rhinitis, syphilitic.....		1	1	1	1
Septum:—										
Deflected.....		26	18	8	17	7	2	26
Gumma.....		1	1	1	1
Spur.....		5	5	5	5
Ulcer.....		1	1	1	1
Sinuses:—										
Frontal, acute inflammation of....		1	1	1	1
Tonsils:—										
Abscess, peritonsillar.....		3	2	1	3	3
Tonsils and Adenoids:—										
Hypertrophy of.....	1	696	369	328	690	2	3	2	697
Turbinate:—										
Cystic middle.....		2	1	1	2	2
	1	773	420	354	740	20	8	3		774
<i>STATISTICAL SUMMARY OF SURGICAL CONDITIONS.</i>										
Total surgical diseases.....	86	1582	847	821	1004	321	124	138	81	1668
Total eye diseases.....	19	298	182	135	143	144	15	2	13	317
Total ear, nose and throat diseases....	1	773	420	354	740	20	8	3	3	774
Total general surgery.....	106	2653	1449	1310	1887	485	147	143	97	2759
Total surgical injuries.....	26	619	505	140	342	166	21	66	50	645
Total surgical cases treated.....	132	3272	1954	1450	2229	651	168	209	147	3404

NOTE.—Of the above number of deaths in the surgical report, sixteen were still-born infants.

EMORY G. ALEXANDER, M.D.,
Surgical Registrar.

GENERAL TABLE OF SURGICAL OPERATIONS.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
ALIMENTARY SYSTEM.						
<i>Appendix:—</i>	Acute appendicitis.....	55	5	60
	Acute appendicitis with abscess.....	20	1	Myocarditis.....	1	22
	Acute appendicitis with abscess and fæcal fistula.....	2	2
	Acute appendicitis with peritonitis.....	21	2	Peritonitis.....	...	23
	Gangrenous appendicitis.....	7	7
	Chronic appendicitis.....	58	2	60
	Chronic appendicitis with intestinal obstruction.....	...	1	{Intestinal obstruction.....}	...	1
	Chronic appendicitis with cystic ovary.....	...	1	Delirium tremens..	...	1
	To cholecystectomy.....	1	1
	To exploratory operation for pericholecystitis...	1	1
Appendectomy incidental to other operations.....	To operations on uterus and appendages.....	49	49
Incision and drainage..	To herniorrhaphy.....	1	1
	Appendicitis with abscess	7	1	Septicæmia.....	...	8
<i>Gums, Alveolæ and Teeth:—</i>	Appendicitis with diffuse peritonitis.....	1	1	{Sepsis and pneumonia.....}	...	2
	Excision.....	2	2
Gall-Bladder and Biliary Passages:—						
Breaking of adhesions..	Pericholecystitis.....	3	3
Cholecystectomy.....	Cholecystitis.....	1	1
	Cholelithiasis.....	7	2	{1 shock..... 1 Pneumonia.....}	...	9
Cholecystostomy.....	Cholecystitis.....	3	3
	Cholelithiasis.....	10	1	Shock.....	2	13
Exploratory.....	Carcinoma of gall bladder	1	1
	Cholecystitis.....	1	1
<i>Hernia:—</i>	Pericholecystitis.....	3	3
	Strangulated inguinal hernia.....	7	1	Peritonitis.....	1	9
Herniotomy and Radical cure.....	Strangulated femoral hernia.....	3	3
	Strangulated ventral hernia of anterior abdominal wall.....	1	1
Radical cure (single)..	Femoral hernia.....	5	5
	Inguinal hernia.....	66	3	69
	Post-operative hernia....	10	10
Radical cure (double)..	Umbilical hernia.....	2	2
	Double inguinal hernia..	9	9
Intestines:—						
Colostomy.....	Intestinal obstruction from adhesions.....	1	1
Entero-enterostomy...	Ruptured intestines.....	1	1
	Fæcal fistula.....	1	1	Shock.....	...	2
Enterorrhaphy.....	Typhoid perforation....	...	3	{1 Intestinal hemorrhage..... 2 Peritonitis.....}	...	3

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
ALIMENTARY SYSTEM (Con.)						
<i>Intestines</i> (Con.):—						
Enterostomy.....	Intestinal obstruction (volvulus)	1	1	Shock.....	1	1
Enterotomy.....	Nail in intestines.....	1	1		1	1
	Adhesions.....	1	1		1	1
Exploratory.....	Carcinoma of colon.....	1	1	Carcinomatosis.....	2	2
	Enteritis, chronic.....	1	1		1	1
	Peritonitis, localized.....	1	1		1	1
	(During typhoid fever)					
	Redundant sigmoid.....	1	1		1	1
Excision (partial).....	To remove gall stones producing intestinal obstruction.....	1	1	Uræmia.....	1	1
Ileo-colostomy.....	Intestinal adhesions.....	1	1	Myocarditis.....	1	1
Laparotomy for.....	Abscess, residual.....	1	1		1	1
	Carcinoma of sigmoid....	1	1		1	1
Laparotomy for and separation of adhesions.....	Intestinal adhesions.....	2	2		2	2
Resection and end to end anastomosis....	Intestinal obstruction by bands, acute.....	1	1		1	1
Sigmoidopexy.....	Prolapse of rectum.....	1	1		1	1
<i>Lip</i> :—						
Excision (radical).....	Epithelioma of lip.....	1	1		1	1
<i>Liver</i> :—						
Exploratory.....	Carcinoma of liver.....	1	1		1	1
Suture.....	Stab wounds.....	1	1		1	1
Talma operation.....	Cirrhosis of liver.....	1	1		1	1
<i>Omentum</i> :—						
Incision and drainage...	Abscess of omentum.....	1	1	{ Cirrhosis of liver and Bright's Disease..... }	1	1
<i>Œsophagus</i> :—						
Œsophagotomy (external)	False teeth in œsophagus	1	1		1	1
Removal of foreign body (through mouth)	{ Coin in œsophagus.....	1	1		1	1
	{ False teeth in œsophagus	1	1		1	1
<i>Pancreas</i> :—						
Cholecystenterostomy. {	Carcinoma head of pancreas.....	1	1		1	1
Cholecystostomy.....	Chronic pancreatitis.....	2	2		2	2
<i>Peritoneum</i> :—						
Excision.....	Abdominal sinus.....	1	1	Acute mania.....	1	1
Laparotomy and drainage.....	Abscess, retroperitoneal..	1	1		1	1
Laparotomy and separation of adhesions...	Adhesions, post-operative	3	3		3	3
<i>Rectum and Anus</i> :—						
Cauterization.....	Prolapse of rectum.....	1	1		1	1
	Hemorrhoids, internal...	22	22		22	22
Clamp and cautery....	Hemorrhoids, combined..	3	3		3	3
	Rectal polypi.....	1	1		1	1
Colostomy.....	Carcinoma of rectum.....	1	1	Shock.....	1	1
Curettage and dilatation of sphincter....	Fissure in ano.....	3	3		3	3

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
ALIMENTARY SYSTEM (Con.)						
<i>Rectum and Anus</i> (Con.):—						
Excision.....	Epithelioma of anus.....	1	1
	Fistula in ano.....	1	1
	Hemorrhoids, external...	1	1
Incision and drainage..	Pilonidal cyst.....	2	2
	Fistula in ano.....	15	15
	Ischio-rectal abscess....	14	14
Ligation.....	Hemorrhoids.....	10	2	12
Resection of rectum (partial).....	Prolapse of rectum.....	1	1
<i>Stomach and Duodenum</i> :—						
Enterorrhaphy.....	Duodenal ulcer, perforated.....	...	1	Peritonitis.....	...	1
Enterorrhaphy and gastroenterostomy..	Duodenal ulcer, perforated.....	1	1
Exploratory.....	Carcinoma of stomach...	1	1
	Gastric ulcer.....	1	1
	Gastric ulcer.....	1	1
Gastrectomy (partial)..	Lympho-sarcoma of stomach.....	1	1
	Carcinoma of stomach with gastroenterostomy	1	1
	Carcinoma of stomach...	...	1	Shock.....	...	1
Gastroenterostomy....	Duodenal ulcer.....	2	2
	Gastric ulcer.....	5	5
	Gastric and duodenal ulcer.....	1	1
Gastroenterostomy with plication of ulcer.....	Perigastric adhesions....	...	1	Shock.....	...	1
	Gastric ulcer.....	1	1
	Hemorrhage from duodenal ulcer.....	1	1
<i>Tongue and Floor of Mouth</i> :—						
Excision (partial).....	Epithelioma of tongue ..	1	1
Incision and drainage..	Epithelioma of tongue with abscess of floor of mouth.....	1	1

ARTICULATIONS.						
Amputation.....	Hammer toe.....	2	2
Aspiration.....	Synovitis of knee, traumatic.....	1	1
	Dislocation of semilunar cartilage.....	4	4
Excision.....	Tuberculosis of wrist....	1	1
	Tuberculous arthritis of knee.....	2	2
Incision and drainage....	Tuberculous arthritis of hip.....	...	1	Septicæmia.....	...	1
	Tuberculous arthritis of hip.....	1	1
Injection of 2% formalin in glycerine.....	Gonococcic arthritis....	4	4
	Synovitis of knee.....	2	2
Osteotomy.....	Genu valgum.....	1	1
Tenotomy.....	Talipes equino-varus	1	1

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
BLOOD-VASCULAR SYSTEM.						
Excision.....	Hæmangioma of foot...	1				1
Phlebectomy.....	Varicose veins of leg.....	6			1	7
FEMALE GENERATIVE ORGANS.						
Amputation.....	Hypertrophied cervix....	2				2
Amputation with cauterization.....	Venereal warts.....		1	Shock.....		1
Cauterization and curettage.....	Carcinoma of anterior vaginal wall.....	2				2
	Carcinoma of cervix.....	1				1
Colporrhaphy (anterior) ..	Cystocele.....	5			1	6
	Abortion, incomplete....	52				52
Dilatation and curettage	Anteflexion of uterus....	1				1
	Endometritis.....	41			1	42
	Menorrhagia.....	1				1
	Puerperal sepsis.....	1				1
	Retained secondines....	7				7
	Stenosis of cervix.....	3				3
Excision.....	Submucous fibroid of uterus.....	1				1
	Epithelioma of vulva ...	1				1
Exploratory.....	Pregnancy.....	1				1
	Pregnancy, extra-uterine, ruptured.....	1				1
Hysterectomy.....	Carcinoma of uterus	1				1
	Epithelioma of cervix....	1				1
(Wertheim.)	Abscess, tubo-ovarian...	2				2
	Carcinoma of uterus	1				1
	Fibroid of uterus.....	6	3	{ 2 Shock..... 1 Peritonitis..... }	1	10
	Endometritis.....	1				1
	Pregnancy, extra-uterine (cornual).....	1				1
	Prolapse of uterus.....	2				2
Hysterectomy.....	Puerperal sepsis.....		1	Sepsis.....		1
	Pyosalpinx, double	1				1
	Salpingitis, acute suppurative.....	1	1	Peritonitis.....		2
	Sarcoma of uterus.....	1				1
	Uterus didelphys	1				1
	Fibroid of uterus with salpingitis.....	1				1
Hysteropexy:—						
Shortening round ligaments (extra-peritoneal).....	Retroversion of uterus...	1				1
	Prolapse of uterus	1				1
Shortening round ligaments (intra-peritoneal).....	Retroflexion of uterus...	2				2
	Retroversion of uterus ..	7				7
Ventro-fixation.....	Prolapse of uterus.....	1				1
	Retroflexion of uterus...	1				1
	Retroversion of uterus...	4				4
	Abscess Bartholin's gland	2			1	3
Incision and drainage....	Abscess, pelvic.	1				1
	Hematoma of vulva.....	1				1
Laparotomy.....	Liberating pelvic adhesions.....	1				1
Laparotomy and drainage	Puerperal sepsis.....		1	Sepsis.....		1
Oophorectomy.....	Ovarian cyst.....	7				7
Perineorrhaphy.....	Lacerated perineum.....	28				28

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
FEMALE GENERATIVE ORGANS (Con.)						
Phlebectomy.....	Varicose veins of broad ligament.....	1	1
Salpingectomy.....	Hydrosalpinx, double....	1	1
Salpingectomy (double) ..	Pregnancy, extra-uterine	1	1
	Salpingitis.....	5	5
	Salpingitis, double.....	1	1
	Adhesions, pelvic.....	1	1
	Abscess, tubo-ovarian...	10	2	12
	Cyst, ovarian.....	2	2
	Cyst, ovarian, calcified..	1	1
Salpingo-oophorectomy (single).....	Hydrosalpinx, double....	1	1
	Pregnancy, extra-uterine	2	2
	Pregnancy, extra-uterine, ruptured.....	7	7
	Salpingo-oophoritis.....	21	21
	Abscess, tubo-ovarian...	4	4
	Abscess, tubo-ovarian, double.....	1	1
Salpingo-oophorectomy (double).....	Cystic ovary.....	1	1
	Hydrosalpinx.....	...	1	Shock.....	...	1
	Pyosalpinx, double.....	1	1
	Pyosalpinx, double, with secondary abscess.....	...	1	Sepsis.....	...	1
	Salpingitis.....	6	6
Snare.....	Uterine polyp.....	2	2
Trachelorrhaphy.....	Lacerated cervix.....	16	16
Wylie drain.....	Endometritis.....	8	8
OBSTETRICAL OPERATIONS						
Cæsarian section.....	Eclampsia.....	...	1	Eclampsia.....	...	1
Cæsarian section (vaginal)	Obstructed labor.....	1	1
Craniotomy.....	Eclampsia.....	...	1	Shock.....	...	1
	Obstructed labor.....	3	3
Forceps.....	Eclampsia.....	1	1
	Obstructed labor.....	3	3
	Placenta prævia.....	1	1
Therapeutic abortion....	Eclampsia.....	1	1
	Pernicious vomiting.....	3	3
Version.....	Malposition.....	5	5
MALE GENERATIVE ORGANS.						
Castration.....	Tuberculous orchitis....	1	1
	Chancroid.....	2	2
Circumcision.....	Paraphimosis.....	2	2
	Phimosis.....	50	1	Heart failure.....	...	51
	Redundant prepuce.....	8	8
Cystostomy, suprapubic..	Hypertrophied prostate .	1	1
Fixation.....	Undescended testicle....	1	1
Phlebectomy.....	Varicocele.....	12	12
Plastic.....	Hypospadias.....	1	1
Prostatectomy, supra-pubic.....	Hypertrophied prostate .	2	3	{ 1 Shock..... 2 Uremia..... }	...	5
Radical cure.....	Hydrocele.....	26	1	27
	Hydrocele of cord, encysted.....	1	1

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
GLANDULAR SYSTEM.						
	Carcinoma of breast.....	1	1
Ablation of breast, radical operation.....	Carcinoma of breast, medullary.....	1	1
	Carcinoma of breast, scirrhus.....	8	8
Excision, plastic.....	Mastitis, chronic fibrous.	1	1
	Cyst of breast.....	1	1
Exploratory.....	Fibro-adenoma of breast	2	2
	Rupture of spleen.....	...	1	Shock.....	...	1
Incision and drainage.....	Mammary abscess.....	2	2
Thyroidectomy.....	Parenchymatous goitre..	2	2
LYMPHATIC SYSTEM.						
Adenectomy.....	Adenitis, cervical.....	12	1	13
	Adenitis, inguinal.....	3	3
Curette and cautery.....	Carcinoma inguinal nodes	...	1	Carcinomatosis....	...	1
	Carcinoma axillary nodes (recurrent).....	1	1
Excision.....	Carcinoma cervical nodes (recurrent).....	1	1
	Sarcoma cervical nodes..	1	1
Exploratory.....	Retroperitoneal sarcoma	1	1
	Adenitis, axillary, suppurative.....	3	3
Incision and drainage....	Adenitis, cervical, suppurative.....	4	4
	Adenitis, inguinal, suppurative.....	3	3
Lymph-angeio-plasty.....	Adenitis, submaxillary, suppurative.....	3	3
	Lymph-œdema of leg....	1	1
NERVOUS SYSTEM.						
Alcoholic injections of trifacial nerve.....	Epithelioma of face.....	3	1	4
	External hydrocephalus..	1	1
Craniotomy.....	Meningo-encephalitis....	1	1
	Chronic leptomeningitis..	1	1
Craniotomy (osteoplastic)	Epilepsy, traumatic.....	1	1
	Fifth nerve (for painful epithelioma).....	1	1
Excision.....	Gunshot wound of vertebræ.....	...	1	Sepsis.....	...	1
	Spastic hemiplegia.....	1	1
Laminectomy.....	Cerebro-spinal fistula....	1	1
OSSEOUS AND CARTILAGINOUS SYSTEM.						
Amputation.....	Dislocation little finger, compound.....	1	1
	Polydactylism.....	1	1
Bone transplantation.....	Supracondylar fracture of humerus, compound	1	1
	Tuberculosis of vertebræ ...	1	...	Pneumonia.....	...	1
Coccygectomy.....	Coccygodnia.....	2	2

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
OSSEOUS AND CARTILAGINOUS SYSTEM (Con.)						
Craniotomy, osteoplastic.	Fracture of vault of skull with extra dural hemorrhage.....	1				1
Curettage.....	Necrosis of tarsal bones..	1				1
Elevation of fragments...	Depressed fracture vault of skull.....		1	Compression.....		1
Examination and application of cast.....	Intracapsular fracture, neck of femur.....	1				1
Excision head of metacarpal.....	Dislocated thumb.....	1				1
Excision of patella and myoplasty.....	Tuberculosis of patella..				1	1
Exploratory laparotomy..	Osteomyelitis of vertebræ...		1	Osteomyelitis.....		1
Extraction.....	Impacted third molar tooth.....	1				1
Fixation by nails.....	Fracture internal epicondyle of humerus...	1				1
	Fracture both bones forearm.....	2				2
	Fracture both bones leg..	1				1
Fixation by plate.....	Fracture femur, subtrochanteric.....	1				1
	Fracture femur, supracondylar.....	2				2
	Fracture tibia.....	1				1
Fixation by suture.....	Fracture patella.....	4				4
	Fracture both patella....				1	1
Fixation by wire.....	Fracture of patella.....	7				7
	Fracture shaft of humerus, compound.....	1				1
	Epiphysitis of radius....	1				1
Incision and drainage....	Fracture of humerus, compound comminuted	1				1
	Necrosis of metacarpal bones.....	1				1
	Periostitis of tibia.....	2				2
	Bone cyst of humerus....	1				1
	Bone cyst of tibia.....	1				1
	Epiphysitis.....	1				1
	Necrosis of astragalus...	1				1
	Necrosis of femur.....	2				2
	Necrosis of maxilla.....	2				2
Incision, curettage and drainage.....	Necrosis of metatarsal...	3				3
	Necrosis of frontal bone.	1				1
	Necrosis of rib.....	2				2
	Necrosis of tarsus.....	1				1
	Necrosis of tibia.....	5				5
	Necrosis of ulna.....	1				1
	Osteomyelitis of tibia....	3				3
Injection of Beck's paste..	Necrosis of tibia, chronic	1				1
Ligation and excision of carotid.....	Carcinoma of maxilla....	1				1
Osteotomy.....	Colles' fracture (old)....	1				1
	Exostosis of os calcis....	1				1
	Dislocation of hip.....	5				5
	Dislocation of shoulder...	5				5
Reduction.....	Fracture both bones leg, compound.....	1				1
	Fracture fifth metacarpal	1				1

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
OSSEOUS AND CARTILAGINOUS SYSTEM (Con.)						
Reduction, open.....	Dislocation of index finger.....	1	1
	Dislocation of thumb....	2	2
	Fracture of surgical neck of humerus, with dislocation of head of humerus.....	1	1
Removal of silver wire....	Necrosis of femur.....	1	1
Sequestrotomy.....	Necrosis of femur.....	1	1
	Necrosis of tibia.....	1	1
Tenotomy and reduction	Fracture tibia and fibula, compound.....	1	1
	Pott's fracture.....	1	1	1
Trephining and curetting.	Osteomyelitis of fibula....	1	1
Trephining and elevation of fragments.....	Osteomyelitis of tibia....	1	1	Septicæmia.....	...	2
	Depressed fracture of skull.....	4	1	Respiratory failure	5
RESPIRATORY SYSTEM.						
Thoracotomy.....	Empyema.....	10	6	{ 4 Shock..... 2 Sepsis..... }	1	17
Tracheorrhaphy and tracheotomy.....	Cut throat.....	...	1	Pneumonia.....	...	1
SOFT PARTS.						
Amputations:—						
Arm.....	Conical stump.....	1	1
	Traumatic amputation of arm.....	2	2
Fingers.....	Crush of finger.....	2	2
	Infected wound of finger.	4	4
Hand.....	Traumatic amputation of finger.....	1	1
	Infected hand.....	1	1	2
	Traumatic amputation of hand.....	1	1
Leg.....	Crush of foot.....	...	1	Shock.....	...	1
	Crush of leg.....	1	1
	Crush of both legs.....	...	1	Shock.....	...	1
	Gangrene of foot, diabetic.....	...	1	Myocarditis.....	...	1
	Gangrene of foot, senile..	...	1	Pneumonia.....	...	1
Shoulder joint.....	Gangrene of leg, senile...	...	1	Pneumonia.....	...	1
	Painful stump.....	1	1
	Traumatic amputation of leg.....	2	2
Toes.....	Tuberculosis of foot.....	1	1
	Lymphœdema of arm.....	1	1
Amputation, cinematoplastic.....	Gangrene of toe.....	4	4
	Sarcoma of toe.....	1	1
Avulsion.....	Traumatic amputation of toe.....	1	1
	Stump of arm.....	1	1
	Ingrowing toe nail.....	1	1

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
SOFT PARTS (Con.)						
Cauterization and curet- tage.....	Epithelioma of nose.....	1	1
	Carbuncle of back.....	1	1
	Carbuncle of knee.....	1	1
	Carbuncle of neck.....	6	1	Sinus thrombosis...	...	7
Excision.....	Carbuncle of shoulder...	1	1
	Ganglion of ankle, com- pound.....	1	1
	Ganglion of wrist.....	3	3
	Branchial cyst.....	1	1
Excision of tumors.....	Ecchondroma of ear.....	1	1
	Epithelioma of eyelid....	1	1
	Epithelioma of face.....	1	1
	Epithelioma of forehead..	1	1
	Epithelioma of nose.....	1	1
	Fibro-lipoma of but- tocks.....	1	1
	Lipoma of back.....	1	1
	Lipoma of buttocks.....	1	1
	Lipoma of shoulder.....	2	2
	Sarcoma of scalp.....	1	1
	Sarcoma of thigh (spindle cell).....	1	1
	Sebaceous cyst of scalp..	1	1
	Abscess, alveolar.....	3	3
	Abscess, arm.....	1	1
	Abscess, abdominal wall..	1	1
Incision and drainage....	Abscess, cervical.....	4	4
	Abscess, maxilla.....	1	1
	Abscess, leg.....	1	1
	Abscess, palmar.....	3	3
	Abscess, popliteal.....	2	2
	Abscess, psoas.....	2	2
	Abscess, thigh.....	1	1
	Cellulitis of forearm.....	2	2
	Cellulitis of leg.....	1	1
	Hematoma, inguinal.....	2	2
	Hematoma of thigh, in- fected.....	1	1
	Infected foot.....	1	1
	Infected hand.....	1	1
Incision, evacuation and closure.....	Psoas abscess.....	1	1
	Gunshot wound of mouth	1	1
Ligation lingual artery....	Bullet in foot.....	2	2
	Bullet in leg.....	1	1
	Bullet in shoulder.....	2	2
	Bullet in skull.....	1	1
Removal of foreign body..	Foreign body in foot.....	3	3
	Foreign body in neck.....	1	1
	Needle in finger.....	1	1
	Needle in hand.....	2	2
Skin grafting.....	Ablation of breast.....	2	2
	Excision of patella.....	1	1
	Lacerated forearm.....	1	1
Suture.....	Laceration of muscles and nerves of forearm..	1	1
Tenorrhaphy.....	Severed tendons.....	3	3
Tenotomy.....	Torticollis.....	2	2
	Spastic hemiplegia.....	1	1

General Table of Surgical Operations—Continued.

OPERATION.	CONDITION FOUND OR PART AFFECTED.	RECOVERED.	DIED.	CAUSE OF DEATH.	REMAINING.	TOTAL.
URINARY SYSTEM.						
Cystostomy, supra-pubic..	Vesical calculus.....	1	1
Cystotomy, supra-pubic. {	Foreign body in bladder..	1	1
	Foreign body in urethra..	1	1
Incision and drainage.... {	Abscess, perinephric	2	2
	Abscess, periurethral....	2	2
Nephrectomy.....	Pyonephrosis.....	1	1
Nephropexy.....	Movable kidney.....	1	1
Nephrotomy.....	Nephrolithiasis.....	3	3
Ureterotomy.....	Ureteral calculus.....	2	2
Urethrotomy, external {	Rupture of urethra.....	1	1
perineal.....	Stricture of urethra.....	5	1
	Foreign body in urethra..	1	5
Urethrotomy, external {	Stricture of urethra.....	2	2
and internal.....	Stricture of urethra.....	2	2
Urethrotomy, internal... {	Stricture of urethra (con-	2	2
	genital).....	1	1
MISCELLANEOUS.						
Intravenous injection of {	Syphilis.....	44	1	45
"606".....						
Totals.....	1251	62	47	1360

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EYE OPERATIONS.

OPERATION.	CONDITION FOUND.	RECOVERED.	DIED.	REMAINING.	TOTAL.
<i>Eyelids:—</i>					
Excision.....	Chalazion.....	1	1
	Cystoid cicatrix.....	1	1
	Epithelioma of eyelid.....	1	1
Plastic operation.....	Ectropion.....	1	1
<i>Conjunctiva:—</i>					
Suturing.....	Lacerated wound of conjunctiva..	1	1
<i>Lachrymal apparatus:—</i>					
Extirpation of sac.....	Dacryocystitis.....	1	1
Incision and drainage.....	Double lachrymal abscess.....	1	1
<i>Cornea:—</i>					
Cauterization.....	Kerato-iritis.....	1	1
Incision.....	Pupillary membrane.....	1	1
Section.....	Bulbous keratitis.....	1	1
<i>Eyeball and orbit:—</i>					
	Buphthalmos.....	1	1
	Glioma of retina.....	1	1
	Hypopyon.....	1	1
	Irido-cyclitis.....	1	1
Enucleation of eyeball.....	Interstitial keratitis with intra-ocular hemorrhage.....	1	1
	Lacerated wound of eyeball.....	1	1
	Panophthalmitis.....	2	2
	Phthisis bulbi.....	2	2
Evisceration.....	Phthisis bulbi.....	1	1
Incision.....	Hypopyon.....	1	1
	Panophthalmitis.....	1	1
Removal by magnet.....	Foreign body in eye.....	7	7
<i>Iris:—</i>					
Iridectomy with extraction of lens.....	Glaucoma, acute.....	2	2
	Iritis, chronic.....	1	1
	Iritis, traumatic.....	5	5
	Senile cataract.....	28	28
	Corneal opacity.....	1	1
Preliminary iridectomy.....	Senile cataract.....	36	36
	Traumatic cataract.....	3	3
Needling.....	Chronic irido-cyclitis.....	1	1
	Capsular cataract.....	1	1
Sclerotomy, anterior.....	Secondary glaucoma.....	1	1
Sclerotomy, posterior.....	Secondary glaucoma.....	1	1
	Acute glaucoma.....	2	2
Trephine.....	Chronic glaucoma.....	1	1
	Secondary glaucoma.....	1	1
<i>Lens and capsule:—</i>					
	Opaque anterior capsule.....	1	1
Capsulotomies.....	Capsular cataract.....	1	1
	Secondary cataract.....	1	1
	Capsular cataract.....	2	2
Discissions.....	Congenital cataract.....	5	5
	Secondary cataract.....	10	10
	Traumatic cataract.....	6	6
Incision.....	Detachment of retina.....	2	2
<i>Muscles:—</i>					
Advancement.....	Strabismus, convergent.....	3	3
	Strabismus, divergent.....	1	1
Tenotomy, external rectus.....	Divergent strabismus.....	2	2
Tenotomy, internal rectus.....	Convergent strabismus.....	8	8
Totals.....		156	156



FIG. 22.—Drug Store.

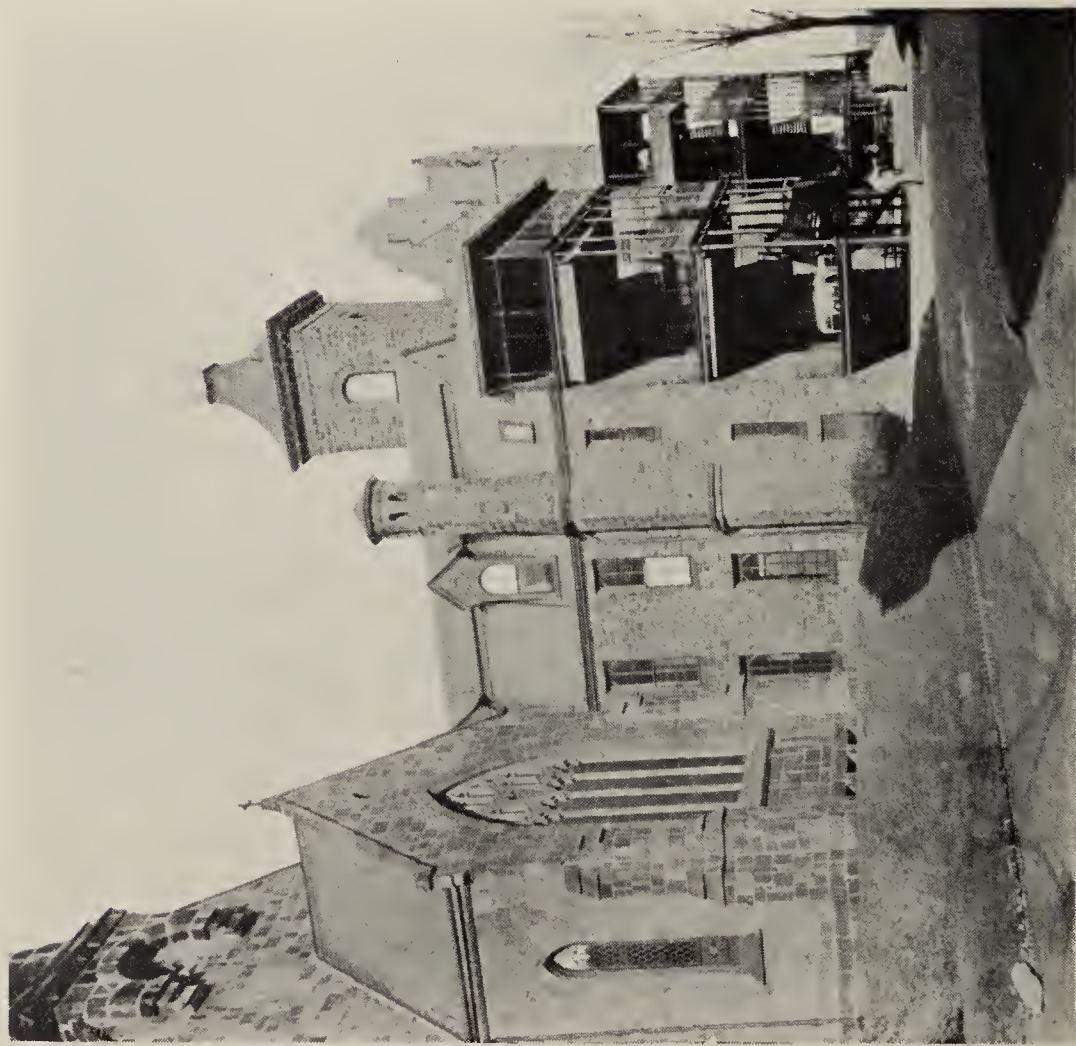


FIG. 23.—Porches at South End of Ward Buildings.



FIG. 24.—Water Tower.

EAR, NOSE, AND THROAT OPERATIONS.

OPERATION.	CONDITION FOUND.	RECOVERED.	DIED.	REMAINING.	TOTAL.
Adenoidectomy and snaring of tonsils.....	Hypertrophied tonsils and adenoids.....	692	2	694
Excision.....	Septal spur.....	4	4
Excision (partial).....	Cystic middle turbinate.....	2	2
Incision and drainage.....	Empyema of maxillary sinus....	1	1
	Mastoid abscess.....	2	2
	Peritonsillar abscess.....	2	2
Mastoidotomy (simple).....	Mastoiditis, acute.....	15	5
Mastoidotomy (simple), with curettment of mastoid sinuses and excision of jugular vein.....	Mastoiditis with thrombosis of lateral sinus.....	1	1
Mastoidotomy (simple), with curettment of mastoid sinuses and ligation of jugular vein.....	Mastoiditis with thrombosis of lateral sinus.....	1	1
Mastoidotomy (radical).....	Mastoiditis.....	2	1	3
Plastic operation.....	Otitis media, suppurative.....	2	2
Snaring.....	Old fracture of nose.....	1	1
	Hypertrophied tonsils.....	1	1
Submucous resection.....	Nasal polyp.....	1	1
	Deflected septum.....	24	24
Totals.....	750	2	2	754

STATISTICS OF OPERATIONS.

OPERATIONS.	RECOVERED.	DIED.	REMAINING.	TOTAL.
Surgical operations.....	1,251	62	47	1,360
Eye operations.....	156	0	0	156
Ear, nose and throat operations.....	750	2	2	754
Total.....	2,157	64	49	2,270

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STATISTICS OF HARRISON MEMORIAL HOUSE.

Diagnosis On Admission.	Year of Admission.	Male.	Female.	Died during 1912, and Cause of Death.	Died.	Transferred to other Institutions during 1912.	Remain- ing.		Total.
							Male.	Female.	
Brain tumor.....	1897	1	1	1
Hemiplegia	1900	1	1	1
"	1903	1	1	1
"	1906	1	Pulmonary congestion..	1	1
"	1907	1	1	1
"	1909	1	1	1
"	1909	1	1	1
"	1910	1	1	1
"	1910	1	1	1
"	1911	1	1	1
Locomotor ataxia....	1897	1	1	1
"	1902	1	1	1
"	1906	1	1	1
"	1909	1	1	1
Posterior sclerosis...	1902	1	1	1
Paraplegia, spastic...	1898	1	1	1
"	1902	1	1	1
"	1905	1	1	1
Paraplegia.....	1899	1	Intestinal obstruction.	1	1
"	1908	1	Exhaustion following hiccoughs ..	1	1
Paraplegia following Pott's disease.....	1908	1	1	1
Paraplegia following fractured vertebræ.	1908	1	1	1
Poliomyelitis.....	1898	1	1	1
"	1903	1	1	1
"	1909	1	1	1
Paralysis, lead	1905	1	1	1
"	1906	1	1	1
Paralysis agitans.....	1911	1	1	1
Huntingdon's chorea.	1907	1	Huntingdon's disease and paresis.	1
Friedrich's ataxia....	1900	1	1	1
Neurasthenia.....	1896	1	1	1
"	1904	1	1	1
Muscular dystrophy..	1898	1	Myocarditis ..	1	1
Senility.....	1900	1	1	1
"	1903	1	1	1
"	1908	1	Pneumonia...	1	1
"	1910	1	1	1
Rheumatism, chronic.	1899	1	Pneumonia...	1	1
"	1910	1	1	1
Rheumatism, muscu- lar.....	1906	1	1	1
Rheumatoid arthritis.	1902	1	1	1
"	1905	1	1	1
"	1909	1	1	1
"	1910	1	1	1
Arthritis deformans.	1904	1	1	1
"	1906	1	1	1



FIG. 25.--Power House and Laundry.



FIG. 26.—Dispensary Building.

STATISTICS OF HARRISON MEMORIAL HOUSE.—Cont'd.

Diagnosis on Admission.	Year of Admission.	Male.	Female.	Died and Cause of Death.	Died.	Transferred to Other Institutions.	Remain- ing.		Total.
							Male.	Female.	
Anthritis deformans...	1911	1	1	1
Ankylosis, hip and spine.....	1907	1	1	1
Chronic endocarditis.	1894	1	1	1
".....	1906	1	1	1
Arterio-sclerosis.....	1905	1	1	1
Chronic interstitial nephritis.....	1898	1	1	1
".....	1906	1	1	1
Chronic bronchitis...	1902	1	1	1
".....	1910	1	1	1
Asthma.....	1909	1	Cardiac dila- tation.....	1	1
Phthisis	1898	1	1	1
Fracture, neck of femur..	1906	1	1	1
	1907	1	1	1
	1908	1	1	1
	1911	1	1	1
Fracture, tibia.....	1908	1	1	1
Pott's disease.....	1899	1	1	1
".....	1910	1	1	1
".....	1911	1	1	1
Fragilitas ossium....	1910	1	1	1
Leg ulcer.....	1909	1	1	1
Supra-pubic and peri- neal fistulæ.....	1903	1	1	1
Enlarged prostate....	1911	1	1	1
Amputation of leg...	1911	1	1	1
Cataracts.....	1903	1	1	1
".....	1906	1	1	1
Paralysis agitans....	1912	1	1	1
Hemiplegia.....	1912	1	1	1
".....	1912	1	1	1
Locomotor ataxia....	1912	1	1	1
Poliomyelitis.....	1912	1	1	1
Senility.....	1912	1	1	1
Recurrent cercinoma of breast.....	1912	1	1	1
Aortic aneurysm.....	1912	1	1	1
Totals.....	38	42	7	I	34	38	80

G. M. ASTLEY, M.D.,
Medical Registrar.

REPORT OF CASES TREATED IN THE MEDICAL DISPENSARY, 1912.

No. of Cases.	No. of Cases.	No. of Cases.
Addison's disease..... 1	Chlorosis..... 1	Gastro-enteritis:—
Adenitis:—	Cholecystitis..... 8	Acute..... 25
Cervical..... 26	Cholangitis..... 4	Chronic..... 1
Inguinal..... 1	Cholelithiasis..... 8	Gastroptosis..... 5
Mediastinal..... 1	Chorea:—	Gingivitis..... 5
Specific..... 1	Habit..... 14	Goitre:—
Submaxillary..... 2	Sydenham's..... 38	Cystic..... 2
Adenoids..... 2	Cirrhosis of liver:—	Exophthalmic..... 10
Adhesions, abdominal... 3	Atrophic..... 5	Simple..... 4
Adiposis..... 1	Diffuse congenital... 1	Gout..... 2
Alcoholism:—	Hypertrophic..... 1	Heart:—
Acute..... 19	Coccygodynia..... 5	Arrhythmia of..... 3
Chronic..... 19	Colic, intestinal..... 2	Bradycardia..... 2
Anæmia:—	Colitis..... 6	Congenital disease of. 1
Chlorotic..... 23	Concussion of brain.... 1	Endocarditis:—
Pernicious..... 26	Constipation:—	Acute..... 10
Secondary..... 49	Acute..... 114	Chronic..... 2
Anal fissure..... 1	Chronic..... 53	Double aortic... 1
Aneurysm of aorta..... 4	Coryza..... 20	Aortic regurgita-
Angio-neurotic edema... 3	Coxalgia..... 3	tion..... 10
Ankylosis..... 1	Cretinism..... 1	Double mitral... 6
Anorexia..... 24	Croup, spasmodic..... 3	Mitral stenosis.. 15
Apoplexy..... 2	Cystitis..... 34	Mitral regurgita-
Appendicitis:—	Debility..... 33	tion..... 97
Acute..... 22	Deformity of chest..... 1	Tricuspid stenosis
Chronic..... 17	Diabetes:—	is..... 1
Recurrent..... 1	Mellitus..... 6	Myocarditis..... 77
Arterio-sclerosis..... 26	Diphtheria (referred to	Tachycardia..... 6
Arthritis:—	family physician).... 6	Heat exhaustion..... 1
Atrophic..... 14	Duodenal ulcer..... 6	Heat cramp..... 1
Deformans..... 8	Dysentery, amoebic... 1	Hemorrhoids:—
Hypertrophic..... 13	Dysuria..... 4	External..... 9
Sacro-iliac..... 4	Edema of face..... 1	Internal..... 10
Traumatic..... 3	Emphysema..... 13	Hemiplegia..... 9
Asthma:—	Enteritis:—	Hepatitis, syphilitic... 1
Cardiac..... 7	Acute catarrhal..... 100	Hernia:—
Bronchial..... 16	Chronic..... 5	Diaphragmatic..... 1
Essential..... 4	Lienteric..... 1	Femoral..... 1
Renal..... 1	Entero-colitis..... 26	Inguinal..... 2
Balanitis..... 2	Enuresis nocturnus.... 35	Umbilical..... 2
Bell's Palsy..... 1	Epilepsy:—	Ventral..... 6
Bronchitis:—	Grand mal..... 12	Hydrocele..... 1
Acute catarrhal..... 697	Jacksonian..... 7	Hydrocele of cord..... 1
Chronic..... 93	Petit mal..... 7	Hypertrophied tonsils.. 32
Sub-acute..... 52	Epistaxis..... 3	Hypertrophied liver... 1
Bursitis..... 1	Erysipelas..... 1	Hyperchlorhydria..... 20
Calculi:—	Fever:—	Hysteria..... 7
Renal..... 1	Typhoid..... 25	Icterus:—
Vesical..... 1	Malarial..... 14	Catarrhal..... 3
Carcinoma:—	Thermic..... 2	Ileo-colitis..... 2
Breast..... 2	Fibroma..... 3	Imbecility..... 5
Gall-bladder..... 1	Fissure in ano..... 1	Incontinence of urine... 3
Clitoris..... 1	Floating kidney..... 3	Incontinence of feces... 1
Face..... 1	Foreign body in stomach 1	Inanition..... 10
Ileo-cæcal..... 1	Furunculosis..... 36	Indigestion..... 21
Jaw..... 1	Gastralgia..... 3	Influenza..... 84
Liver..... 2	Gastric ulcer..... 22	Insomnia..... 4
Pylorus..... 3	Gastritis:—	Jaundice, acute catarrhal 3
Rectum..... 4	Acute catarrhal..... 237	Laryngitis:—
Stomach..... 3	Alcoholic..... 34	Acute catarrhal..... 27
Tongue..... 1	Chronic..... 48	Chronic..... 2
Caries of teeth..... 3	Hyperacidity..... 75	Syphilitic..... 1
Cephalalgia..... 35	Sub-acute..... 5	Lumbago..... 135
Cephalalgia, septic.... 1	Gastrodynia..... 1	Malingering..... 9
Chancroids..... 1		Malnutrition..... 1

MEDICAL DISPENSARY—Continued.

No. of Cases.		No. of Cases.		No. of Cases.	
Mania, acute.....	3	Pharyngitis—Con.:—		Torticollis.....	7
Marasmus.....	16	Syphilitic.....	4	Urethral stricture.....	2
Measles.....	7	Tuberculous.....	1	Urethritis, specific.....	41
Metatarsalgia.....	1	Phimosis.....	3	Uremia.....	1
Mastoiditis.....	3	Phlebitis.....	2	Vaccination.....	2
Masturbation.....	1	Phthisis:—		Varicella.....	4
Melancholia.....	3	Acute pulmonic.....	41	Varicose ulcers.....	3
Meningitis.....	1	Chronic ulcerative...	30	Varicose veins.....	2
Migraine.....	8	Fibroid.....	12		
Myalgia.....	44	Glandular.....	5	Vermes:—	
Myelitis, anterior-polio.	7	Incipient pulmonic....	20	Ascaris lumbricoides..	1
Myositis.....	22	Laryngeal	1	Oxyuris vermicularis..	6
Nephritis: —		Pleurisy:—		Tænea saginata.....	10
Acute parenchyma-		Acute.....	46	Tænea solium.....	5
tous.....	10	Chronic.....	23	Verruca.....	2
Chronic interstitial...	22	Effusive.....	8	Vertigo.....	11
Chronic parenchyma-		Pleurodynia.....	69	Vesical calculus.....	1
tous.....	3	Plumbism.....	17	Convalescent from	
Nephrolithiasis.....	8	Pneumonia:—		house.....	8
Nephroptosis.....	1	Croupous.....	7	Refused admission.....	11
Neuralgia:—		Podalgia.....	2	Refused treatment.....	25
Intercostal.....	22	Pott's Disease.....	2	Referred to dentist....	2
Occipital.....	4	Prolapse of rectum....	4	Referred to family	
Post tibial.....	1	Prostatic hypertrophy..	11	doctor.....	30
Supra-orbital.....	14	Prostatitis.....	1	Transferred to Eye Dis-	
Trifacial.....	13	Pruritus:—		pensary.....	42
Neurasthenia:—		Ani.....	4	Transferred to Ear,	
Alcoholic.....	36	Hands	1	Nose and Throat Dis-	
Idiopathic.....	78	Vulvæ.....	1	pensary.....	149
Post-operative.....	14	Purpura hemorrhagica .	2	Transferred to Surgical	
Post-puerperal.....	9	Purpura rheumatica ...	1	Dispensary.....	134
Sexual.....	11	Pyelitis.....	1	Transferred to wards...	253
Neuritis:—		Pyorrhoea alveolaris...	1	Undiagnosed.....	172
Alcoholic.....	5	Rachitis.....	3		
Brachial.....	7	Ranula	1	Diseases of the Skin.	
Circumflex.....	5	Retention of urine.....	1	Acne:—	
Intercostal.....	1	Rheumatism:—		Rosacea.....	3
Multiple.....	2	Acute articular.....	118	Pustular.....	6
Musculo-spiral.....	7	Chronic articular....	74	Vulgaris.....	3
Sciatic.....	16	Gonorrhœal.....	6		
Occupation.....	3	Muscular.....	111	Dermatitis:—	
Supra-orbital.....	1	Rheumatoid arthritis...	2	Erythema.....	8
Traumatic.....	2	Rhinitis.....	7	Exfoliative.....	2
Ulnar.....	1	Scarlatina	2	Herpetiformis.....	1
Tibial.....	3	Sciatica.....	16	Venenata.....	7
No disease.....	4	Sclerosis:—		Vesicular.....	1
Odontalgia	2	Post Lateral.....	2		
Orchitis.....	2	Scoliosis.....	1	Eczema:—	
Osteo-periostitis.....	1	Senility.....	7	Acute erythematous..	20
Osteo-periostitis, trau-		Singultus.....	2	Acute vesicular.....	31
matic.....	1	Sinusitis, frontal.....	1	Fissured.....	10
Otitis media.....	5	Spastic hemiplegia....	1	Papular.....	13
Paralysis:—		Splanchnoptosis.....	4	Pustular.....	12
Agitans.....	1	Sprain of back	1	Rubrum.....	6
Birth	1	Stomatitis:—		Seborrhœic.....	2
Infantile.....	3	Apthous.....	5	Squamous.....	18
Paresis.....	2	Catarrhal.....	5		
Paresthesia.....	3	Ulcerative.....	9	Erythema:—	
Parotitis.....	22	Syphilis:—		Multiforme.....	4
Pericarditis.....	5	Congenital.....	3	Nodosum.....	1
Pertussis (referred to		Hereditary.....	1		
family physician)....	12	Primary.....	6	Herpes:—	
Pes planus.....	25	Secondary.....	28	Labialis.....	3
Pes varus.....	1	Tertiary.....	11	Simplex.....	4
Pharyngitis:—		Tenosynovitis	2	Zoster.....	9
Acute.....	29	Tonsillitis:—		Impetigo contagiosa....	37
Chronic.....	9	Follicular.....	136	Intertrigo.....	1
		Suppurative.....	15		
				Pediculosis:—	
				Capitis	9
				Corporis	4

MEDICAL DISPENSARY—Continued.

No. of Cases.	No. of Cases.	No. of Cases.
<i>Diseases of the Skin—Con.</i>	Abortion:—	Ovarian cyst..... 2
Pemphigus..... 1	Incomplete..... 11	Ovaritis..... 1
Pityriasis rosea..... 6	Threatened..... 3	Pregnancy, ectopic..... 1
Psoriasis..... 17	Amenorrhoea..... 20	Pregnancy, normal..... 51
Rhus toxicodendron.... 28	Carcinoma of cervix.... 1	Salpingitis..... 37
Scabies..... 63	Carcinoma of uterus.... 4	Stenosis of cervix..... 2
	Cystocele..... 5	Retained secundies..... 3
	Dysmenorrhoea..... 7	Suppressed menses.... 2
Tinea:—	Endometritis..... 19	Uterus:—
Circinata..... 6	Laceration of:—	Anteflexion..... 3
Tonsurans..... 2	Cervix..... 10	Fibroid of..... 5
Urticaria..... 23	Perineum..... 16	Prolapse of..... 5
	Leucorrhoea..... 8	Retroflexion..... 5
	Mastitis..... 3	Retroversion..... 8
<i>Diseases of Women.</i>	Menopause..... 9	Vaginitis..... 7
Adenoma of breast.... 1	Menorrhagia..... 14	Vulvitis..... 2
	Metrorrhagia..... 11	Total..... 6071



FIG. 27.—Room in Surgical Dispensary.

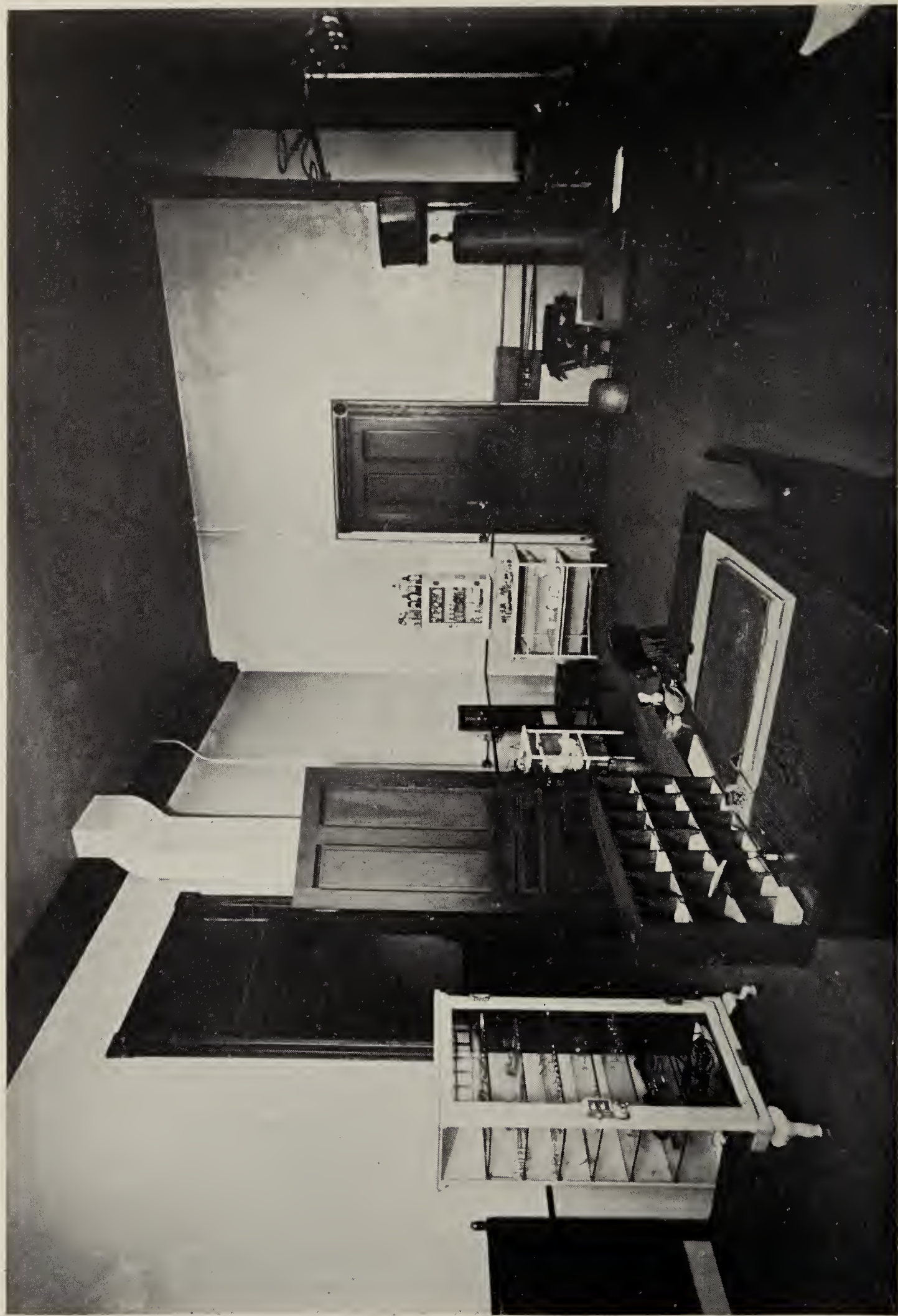


FIG. 28.—Ear, Nose, and Throat Dispensary.

REPORT OF CASES TREATED IN THE SURGICAL DISPENSARY, 1912.

No. of Cases.	No. of Cases.	No. of Cases.
Abrasions..... 93	Bites:—	Endometritis..... 1
Abscess:—	Cat..... 1	Enlarged turbinate... 2
Alveolar..... 32	Dog..... 95	Enuresis..... 1
Arm..... 25	Frost..... 29	Epilepsy..... 5
Axillary..... 17	Horse..... 5	Epiphyseal separation. 19
Breast..... 6	Human..... 11	Epiphysitis..... 1
Buttocks..... 7	Insect..... 5	Epistaxis..... 4
Cervical..... 44	Monkey..... 1	Epulis..... 2
Chest..... 5	Rat..... 1	Erysipelas..... 2
Ear..... 5	Bronchitis..... 3	Exostosis..... 6
Eye-brow..... 2	Bunions..... 5	Fissure in ano..... 6
Face..... 28	Burns:—	Fistula in ano..... 8
Finger..... 36	Acid..... 11	Fistula on face..... 1
Foot..... 12	Alkali..... 5	Foreign bodies:—
Hand..... 23	Brush..... 16	Arm..... 8
Head..... 9	Electric..... 32	Ear..... 3
Hip..... 2	Fat..... 2	Esophagus..... 7
Inguinal..... 12	Fire..... 171	Eye..... 130
Ischio-rectal..... 8	Iodine..... 3	Finger..... 43
Jaw..... 7	Lime..... 2	Foot..... 13
Knee..... 6	Metal..... 8	Hand..... 45
Leg..... 23	Oil..... 1	Head..... 2
Lip..... 2	Pitch..... 1	Intestines..... 1
Mastoid..... 4	Powder..... 12	Leg..... 4
Neck..... 5	Scalds..... 70	Nose..... 1
Nose..... 1	Sun..... 3	Pharynx..... 3
Palmar..... 24	Bursitis..... 31	Stomach..... 1
Parotid..... 7	Calculi:—	Urethra..... 3
Pelvic..... 1	Renal..... 1	Fractures:—
Penis..... 1	Vesical..... 2	Compound:—
Perineal..... 4	Callus..... 6	Femur..... 1
Peritonsillar..... 2	Carbuncle..... 28	Finger..... 12
Popliteal..... 1	Carious teeth..... 42	Humerus..... 5
Psoas..... 3	Caruncles, urethral... 1	Malar..... 2
Pulmonary..... 1	Cellulitis..... 51	Mandible..... 4
Scalp..... 13	Cholelithiasis..... 1	Metacarpal..... 7
Scrotum..... 1	Chorea..... 1	Nasal..... 2
Submaxillary..... 1	Clavus..... 2	Phalanx..... 1
Tubo-ovarian..... 1	Coccygodynia..... 2	Radius..... 2
Thigh..... 1	Concussion..... 5	Radius and ulna.. 8
Vulvar..... 4	Conjunctivitis..... 6	Tibia..... 2
Wrist..... 10	Constipation..... 3	Toe..... 6
Adenitis:—	Contractures from burns..... 5	Simple:—
Axillary..... 19	Contusions:—	Carpal..... 8
Cervical..... 49	Head and face..... 79	Clavicle..... 59
Inguinal..... 19	Lower extremity... 279	Coccyx..... 1
Submaxillary..... 5	Trunk..... 177	Colles'..... 150
Adhesions..... 3	Upper extremity... 279	Femur..... 12
Alcoholism..... 16	Cystitis..... 6	Fibula..... 3
Aneurysm, traumatic.. 1	Deformity of toe..... 2	Malleolus, exter- nal..... 4
Anthrax..... 1	Duhring's disease.... 1	Malleolus, inter- nal..... 1
Antitoxin injection... 4	Duodenal ulcer..... 1	Humerus:—
Arthritis:—	Dupuytren's contrac- ture..... 3	Anatomical neck..... 5
Acute..... 20	Edema:—	Epicondyle.... 1
Chronic..... 13	Legs..... 4	External con- dyle..... 10
Hypertrophic..... 17	Penis..... 2	Intercondyloid.. 5
Suppurative..... 1	Scrotum..... 1	Internal con- dyle..... 11
Gonococcic..... 1	Prepuce..... 1	
Traumatic..... 5	Electric shock..... 1	
Tuberculous..... 13	Empyema..... 7	
Avulsion of:—		
Fingernail..... 9		
Toenail..... 10		

SURGICAL DISPENSARY—Continued.

No. of Cases.	No. of Cases.	No. of Cases.
Fracture (Con.):—	Indigestion..... 2	Poliomyelitis, anterior. 4
Simple (Con.):—	Ingrowing toenail.... 37	Potts' disease..... 4
Humerus (Con.):	Laryngitis..... 2	Pregnancy..... 4
Shaft..... 27	Leucorrhœa..... 4	Prolapse of rectum.... 2
Supracondylar. 6	Lumbago..... 9	Prostatic enlargement. 4
Internal and ex-	Luxations:—	Pyorrhœa alveolaris... 1
ternal con-	Ankle..... 5	Pyosalpinx..... 1
dyle, with	Clavicle..... 9	Renal colic..... 2
fracture of	Elbow..... 12	Retention of urine.... 1
olecranon	Finger..... 22	
process..... 2	Mandible..... 3	Rheumatism:—
Surgical neck... 12	Patella..... 1	Acute..... 10
"T" fracture... 8	Radius..... 6	Chronic..... 9
Ilium..... 6	Sacro-iliac joint.... 3	Rickets..... 1
Maxilla, inferior.. 5	Semilunar cartilage.. 9	
Metacarpal..... 51	Shoulder..... 24	Rupture:—
Metatarsal..... 23	Thumb..... 11	Artery..... 1
Nose..... 18	Ulna..... 4	Eyeball..... 1
Olecranon..... 8	Lymphangitis..... 7	Muscles..... 3
Os calcis..... 1	Malaria..... 1	Tendons..... 3
Patella..... 8	Mastitis..... 7	Vessels..... 2
Phalanx..... 47	Mastoiditis..... 4	Salpingitis..... 7
Pott's..... 15	Masturbation..... 1	Scapulodynia..... 1
Radius..... 50	Miscarriage..... 2	Sciatica..... 4
Ribs..... 66	Mitral regurgitation... 2	Scoliosis..... 2
	Myalgia..... 6	Sinus of scalp..... 1
	Myositis..... 3	Sinus, post-operative.. 2
Scapula:—	Necrosis:—	Skin diseases:—
Acromion pro-	Femur..... 4	Acne rosacea..... 2
cess..... 3	Mandible..... 2	Acne vulgaris..... 2
Body..... 3	Os calcis..... 1	Dermatitis..... 21
Scaphoid..... 1	Phalanx..... 4	Eczema:—
Skull..... 6	Ribs..... 2	Fissum..... 6
Tarsus..... 3	Tarsus..... 2	Pustulosum..... 16
Tibia..... 15	Tibia..... 6	Rubrum..... 15
Ulna..... 16	Nephritis, chronic.... 1	Seborrhœic..... 3
Radius and ulna.. 45	Nephrolithiasis..... 2	Squamosum..... 56
Tibia and fibula... 9	Neuralgia..... 7	Vesiculosum..... 4
Furuncle..... 116	Neurasthenia..... 5	Erythema nodosum. 1
Ganglion..... 26	Neuritis..... 14	Erythema multi-
Gangrene..... 2	Odontalgia..... 15	forme..... 1
Gastritis..... 6	Osteomyelitis..... 6	Herpes:—
Genu valgum..... 1	Osteoperiostitis..... 3	Facialis..... 2
Gingivitis..... 4	Otitis media..... 2	Labialis..... 1
Glossitis..... 1	Painful stump..... 1	Zoster..... 10
Gout..... 1		Impetigo contagiosa 63
Goitre..... 2	Palsy:—	Lupus vulgaris..... 2
Hæmoptysis..... 1	Brachial..... 2	Pediculosis:—
Hallus valgus..... 1	Bell's..... 2	Capitis..... 4
Hammer-toe..... 3	Paralysis:—	Corporis..... 2
Hematoma..... 5	Circumflex..... 1	Pubis..... 1
Hemophilia..... 1	Musculo-spiral..... 7	Pruritus..... 3
Hemorrhage, second-	Perineal nerve..... 1	Psoriasis..... 2
ary..... 1	Ulnar..... 1	Purpura rheumat-
Hemorrhoids:—	Paronychia..... 21	ica..... 2
External..... 10	Parotitis..... 10	Scabies..... 24
Internal..... 10	Periarthritis..... 1	Scrofuloderma.... 1
Combined..... 5	Perichondritis..... 1	Seborrhœa..... 3
Hernia:—	Periostitis..... 13	Tinea-circinata... 11
Incisional..... 4	Pes planus..... 22	Tinea-sycosis..... 1
Inguinal..... 41	Pharyngitis..... 3	Tinea-versicolor.. 2
Umbilical..... 6	Phimosis..... 55	Tinea-tonsurans.. 6
Ventral..... 3	Phlebitis..... 3	Urticaria..... 4
Hordeolum..... 2	Pleurisy..... 5	Verruca..... 8
Hydrocele..... 12	Poisoning:—	
Hyperidrosis..... 1	Gas..... 1	Sprains:—
Hypospadias..... 1	Iodine..... 2	Lower extremity.... 162
Impacted molar..... 1	Lead..... 1	Trunk..... 60
Inflamed heel..... 1	Rhus toxicodendron 60	Upper extremity.... 267



FIG. 29.—Superintendent's House.



FIG. 30.—One of the Open Air Wards for Tuberculous Patients.

SURGICAL DISPENSARY—Continued.

No. of Cases.	No. of Cases.	No. of Cases.
Strain:—	Venereal diseases:—	Lacerated:—
Muscles..... 2	Arthritis, gonococcic..... 4	Cervix..... 21
Stomatitis..... 5	Bartholinitis..... 1	Head and face.... 766
Synovitis..... 26	Chancroid..... 13	Lower extremity.. 181
Supernumerary toes... 1	Condylomata..... 4	Palate..... 1
Talipes equino-varus.. 3	Epididymitis..... 5	Perineum..... 1
Talipes planus..... 2	Orchitis..... 7	Tongue..... 11
Tenosynovitis..... 38	Stricture..... 12	Trunk..... 12
Testicle, undescended. 1		Upper extremity.. 842
Thecitis..... 3		
Tongue-tie..... 3	Syphilis:—	Punctured:—
Tonsillitis..... 8	Primary..... 17	Head and face.... 15
Torticollis..... 7	Secondary..... 20	Lower extremity.. 95
Tuberculosis, pulmonary..... 6	Tertiary..... 19	Trunk..... 6
Tumors:—	Urethritis, gonococcic..... 84	Upper extremity.. 62
Benign:—	Vaginitis..... 4	Stab..... 16
Fibroma..... 4	Verruca..... 13	
Hemangioma..... 1	Vertigo..... 10	Traumatic amputation:—
Keloid..... 1		Finger..... 83
Lipoma..... 6	Wounds:—	Arm..... 1
Nevus..... 3	Crushed:—	Toe..... 10
Papilloma..... 9	Arm..... 8	
Polypi..... 3	Finger..... 62	Cases referred to:—
Cysts:—	Foot..... 9	Ear, Nose and Throat Dispensary 31
Chalazion..... 5	Hand..... 9	Eye Dispensary.... 39
Dermoid..... 1	Neck..... 1	Wards..... 47
Ovarian..... 2	Thumb..... 7	Dentist..... 6
Sebaceous..... 34	Toe..... 7	District Physician... 2
Thyro-glossal..... 2	Gunshot..... 15	Medical Dispensary. 57
Malignant:—	Incised:—	Cases of teeth extraction..... 169
Carcinoma..... 18	Head and face.... 17	Convalescent from Wards..... 87
Epithelioma..... 14	Lower extremity.. 6	Diagnosis unclassified.. 155
Sarcoma..... 4	Trunk..... 4	Eloped..... 4
Ulcers..... 68	Upper extremity.. 31	Examination negative. 17
Uterus:—	Infected:—	Refused treatment.... 4
Retroversion..... 1	Head and face.... 48	
Vaccination..... 557	Lower extremity.. 173	Total..... 9490
Varicocele..... 7	Trunk..... 9	
Varicose ulcers..... 58	Upper extremity.. 492	
Varicose veins..... 33		

REPORT OF CASES TREATED IN THE EAR, NOSE, AND THROAT DISPENSARY, 1912.

No. of cases.	No. of cases.	No. of cases.
<i>Nose and Throat.</i>	<i>Ear Cases.</i>	<i>Septum:—</i>
Referred:—	Affections external ear:—	Deflected..... 93
Dentist..... 5	Cerum, impacted.... 165	Hematoma..... 4
Eloped..... 22	Eczema..... 13	Abscess..... 2
Examination negative 25	Foreign bodies im-	Spur..... 26
Eye Dispensary..... 13	pacted..... 4	Ulcer..... 13
Medical Dispensary.. 24	Furuncle..... 62	Fracture..... 4
Surgical Dispensary.. 19	Polypi..... 41	Sinusitis, antrum..... 2
Total..... 108	Perforation of drum.. 1	Sinusitis, ethmoidal.... 9
	Contused auditory	Sinusitis, frontal..... 5
	canal..... 1	Total..... 600
	Otitis externa..... 15	
	Affections middle ear:—	<i>Throat Cases.</i>
	Mastoiditis..... 11	Æsophagus, stricture of. 1
	Myringitis..... 8	Gingivitis..... 3
	Otitis media:—	Laryngeal paralysis.... 7
	Acute catarrhal... 17	Laryngitis:—
	Chronic catarrhal. 155	Acute..... 13
	Acute suppurative. 139	Chronic..... 14
	Chronic suppurative..... 119	Specific..... 1
	Total..... 720	Tuberculous..... 7
		Aphonia, hysterical.... 1
	<i>Nose Cases.</i>	Peritonsillar abscess.... 26
	Abrasions and contu-	Pharyngitis:—
	sions..... 4	Acute..... 49
	Herpes nasalis..... 1	Chronic..... 13
	Cellulitis..... 6	Granular..... 10
	Epistaxis..... 1	Mycosis..... 1
	Foreign body..... 1	Specific..... 5
	Hay fever..... 2	Stomatitis..... 3
	Ethmoiditis, purulent.. 1	Thyroid enlargement... 7
	Polypi..... 17	Tonsillitis:—
	Rhinitis:—	Acute simple..... 8
	Acute..... 22	Acute follicular..... 73
	Atrophic..... 47	Diphtheria..... 6
	Hypertrophic..... 131	Chronic..... 1
	Pseudo-membranous. 2	Total..... 249
	Purulent..... 19	Total number new cases
	Sclerotic..... 180	treated..... 2576
	Specific..... 7	
	Synechia..... 1	
<i>Operations.</i>		
Polypi:—		
Aural..... 9		
Nasal..... 16		
Spurs removed..... 15		
Peritonsillar abscesses		
incised..... 10		
Removal of tonsils.... 3		
Deflected septum..... 36		
Incision drum mem-		
brane..... 2		
Removal middle tur-		
binal..... 3		
Incision of septal ab-		
cess..... 4		
Drainage antrum of		
Highmore..... 1		
Removal of lingual ton-		
sils..... 2		
Adenoids..... 27		
Faucial tonsils..... 26		
Lingual tonsils..... 25		
Tonsils and adenoids... 717		
Empyema antrum..... 3		
Total..... 899		

REPORT OF CASES TREATED IN THE EYE DISPENSARY, 1912.

No. of cases.	No. of Cases.	No. of Cases.
Abscess..... 13	Palsies:—	Keratitis:—
Burns..... 9	Bell's..... 3	Chronic..... 6
Cellulitis..... 2	Third nerve, diphther- itic..... 2	Dendritic..... 3
Blepharitis..... 124	Fifth nerve, following extirpation Gasse- rian ganglion..... 1	Fascicular..... 2
Chalazion..... 96	Sixth nerve, diphther- itic..... 1	Hypopyon..... 8
Suppurative..... 8	Strabismus:—	Infected..... 3
Cyst, sebaceous..... 6	Convergent:—	Interstitial..... 14
Distichiasis..... 12	Left..... 54	Marginal..... 58
Ecchymosis..... 22	Right..... 49	Pannus..... 2
Edema..... 9	Alternating..... 10	Phlyctenular..... 39
Ectropion..... 5	Concomitant, inter- nal..... 17	Recurrent ulcerative..... 32
Eczema..... 17	Divergent:—	Sclerosing..... 1
Entropion..... 4	Left..... 11	Specific..... 1
Epicanthus, traumatic.. 1	Right..... 7	Striped..... 1
Epithelioma..... 2	Alternating..... 3	Superficial..... 8
Erosion of lids..... 1	Total..... 257	Tuberculous..... 6
Foreign body beneath skin of eyebrow..... 1	<i>Affections of Conjunctiva.</i>	Traumatic..... 35
Hordeolum, suppurative 76	Burns:—	Ulcerative..... 45
Herpes zoster..... 2	Acid..... 5	Vascular..... 8
Ivy poisoning..... 1	Alkaline..... 4	Leucoma..... 14
Papilloma of caruncle.. 1	Lime..... 4	Anterior adherent... 4
Pediculosis ciliaris..... 2	Powder..... 6	Maculae..... 41
Ptoxis..... 1	Conjunctivitis:—	Nebulae..... 29
Scleracedema..... 1	Acute catarrhal..... 269	Ulcer of cornea, central. 10
Symblepharon..... 2	Calcareous..... 1	Deep infected..... 6
Trachoma..... 1	Chronic catarrhal... 104	Infiltrated..... 1
Trichiasis..... 2	Follicular..... 23	Marginal..... 4
Verruca..... 2	Granular..... 8	Simple..... 33
Wounds:—	Kerato..... 2	Traumatic..... 20
Burns..... 2	Phlyctenular..... 66	Cystoid cicatrix..... 1
Contused..... 9	Pseudo-membranous. 1	Staphyloma of cornea.. 1
Infected..... 1	Purulent..... 20	Wounds:—
Lacerated..... 12	Retrotarsal..... 17	Abrasions..... 36
Total..... 447	Subacute catarrhal... 87	Contused..... 1
<i>Affections of Lachrymal Apparatus.</i>	Traumatic..... 41	Incised..... 5
Dacryocystitis..... 31	Vernal..... 2	Infected..... 2
Lachrymal abscess..... 7	Chemosis of conjunctiva 9	Lacerated..... 1
Lachrymal catarrh..... 5	Ecchymosis..... 8	Penetrating..... 7
Obstruction of Duct:—	Irritable stumps..... 2	Perforated..... 8
Double..... 17	Foreign body..... 17	Sclero-keratitis..... 5
Single..... 27	Papilleoma..... 1	Total..... 847
Prelachrymal abscess... 1	Pingucula..... 3	<i>Affections of Eyeball and Orbit.</i>
Total..... 88	Subconjunctival hem- orrhage..... 6	Abscess of orbit..... 1
<i>Nervous and Muscular Affections.</i>	Trachoma..... 3	Cyst of ethmoid..... 1
Amblyopia:—	Wounds:—	Cyst of orbit..... 1
Exanopsia..... 2	Contused..... 2	Fracture of orbit..... 1
Toxic..... 11	Infected..... 3	Frontal sinusitis..... 1
Atrophy of optic nerve, double..... 29	Lacerated..... 4	Glaucoma:—
Atrophy of optic nerve, partial..... 1	Penetrating..... 3	Acute..... 5
Habit chorea..... 5	Total..... 721	Absolute..... 1
Mydriasis, accidental... 3	<i>Affections of Cornea and Sclera.</i>	Chronic..... 7
Nictitation..... 17	Burns:—	Double..... 1
Nystagmus..... 11	Chemical..... 9	Secondary..... 3
Optic neuritis..... 10	Electric..... 1	Orbital cellulitis..... 1
Proptosis..... 2	Fire..... 2	Ophthalmia neonatorum 2
Ptoxis..... 5	Metal..... 3	Panophthalmitis..... 1
Paralysis:—	Conical cornea..... 1	Phthisis bulbi..... 5
External rectus..... 2	Episcleritis..... 19	Foreign body..... 16
Superior rectus..... 1	Foreign body..... 311	Irritable stump follow- ing enucleation..... 1
		Pterygium..... 3
		Wounds:—
		Contused..... 21
		Lacerated..... 1
		Penetrating..... 2
		Total..... 75

EYE DISPENSARY—Continued.

No. of Cases.	No. of Cases.	No. of Cases.
<i>Affections of Uveal Tract.</i>		
Arterio-sclerosis..... 1	Foreign body in iris.... 1	Myopic astigmatism
Choroideremia..... 4	Foreign body in vitreous 4	combined with myopia and presbyopia. 14
Choroiditis:—	Neuro-retinitis..... 10	Presbyopia..... 46
Atrophic..... 6	Total..... 239	Presbyopia combined
Disseminated..... 12		with myopia..... 7
Macular..... 9	<i>Affections of Lens.</i>	Paralysis of accommodation..... 1
Congenital coloboma of iris..... 4	Aphakia..... 12	Total..... 1726
Cyclitis..... 4	Cataract:—	Eloped..... 35
Descemætitis..... 2	Anterior polar..... 4	Referred to other dispensaries..... 20
Edema..... 2	Calcareous..... 9	Total new cases... 4618
Eccentric pupil..... 1	Capsular..... 4	
Embolism central artery of retina..... 1	Complicated..... 3	
Hyalitis..... 2	Congenital..... 3	
Irido-cyclitis..... 4	Cortical..... 2	
Irido-dialysis..... 2	Incipient..... 34	
Iritis:—	Secondary..... 5	
Acute plastic..... 21	Posterior polar..... 5	
Acute specific..... 7	Senile..... 68	
Chronic..... 12	Traumatic..... 12	
Traumatic..... 5	Luxation of lens..... 2	
Kerato-iritis..... 23	Total..... 163	
with hypopyon..... 1		
Paralysis of iris..... 2	<i>Errors of Refraction and Accommodation.</i>	
Partial coloboma of iris 1	Anisometropia..... 3	
Prolapse of iris..... 4	Asthenopia, accommodative..... 107	
Retained pupillary membrane..... 1	Asthenopia, nervous.... 12	
Retinal detachment..... 8	Hypermetropia..... 67	
Retinal hyperæsthesia.. 2	Combined with presbyopia..... 39	
Retino-choroiditis..... 23	Hypermetropic astigmatism..... 64	
Traumatic..... 1	Hypermetropic astigmatism combined with hypermetropia..... 892	
Retinitis:—	Hypermetropic astigmatism combined with hypermetropia and presbyopia..... 261	
Albuminuric..... 2	Mixed astigmatism..... 46	
Chronic..... 4	Myopia..... 38	
Diabetic..... 1	Myopic astigmatism.... 22	
Hemorrhagic..... 7	Myopic astigmatism combined with myopia..... 107	
Macular..... 1		
Pigmentosa..... 4		
Specific..... 5		
Rupture of choroid..... 1		
Sarcoma of choroid..... 1		
Synchysis scintillans... 1		
Uveitis..... 4		
Vitreous opacities..... 27		
Vitreous hemorrhage... 1		

REFRACTION REPORT.

Accommodative asthenopia.....	5
Anisometropia.....	43
Hyperopia.....	208
Hyperopia combined with hyperopic astigmatism.....	489
Hyperopia with hyperopic astigmatism and presbyopia.....	73
Hyperopia with presbyopia.....	66
Hyperopic astigmatism.....	25
Hyperopic astigmatism with presbyopia.....	2
Mixed astigmatism.....	63
Mixed astigmatism with presbyopia.....	11
Myopia.....	33
Myopia combined with myopic astigmatism.....	88
Myopia with myopic astigmatism and presbyopia.....	9
Myopia with presbyopia.....	10
Myopic astigmatism.....	13
Myopic astigmatism with presbyopia.....	3
Presbyopia.....	80
Total number of patients refracted.....	1221

REPORT OF THE PATHOLOGICAL LABORATORY.

THE following report of the Pathological Laboratory for the year 1912 shows a total of 21,111 examinations. This is an increase of 2700 examinations over the previous year. Increases of nearly 3000 requests yearly have been the rule for the past five years, and, in itself, shows the general usefulness of the Laboratory to the clinician in his examinations in the wards.

The Laboratory quarters, completed over a year ago, have proven comfortable and serviceable in every way, and well located for the work required of them.

The Resident service, consisting of a Senior and Junior Resident Physician, continues as in the previous year. In the coming year the reorganization of the Resident Staff of physicians, whereby the Junior Resident Physician of each ward will be required to complete the laboratory work for the cases in that ward, is looked forward to with much interest. Under this new arrangement the Laboratory will be provided with a Special Resident Physician, who will continue on service one year. In the past, the increasing number of Laboratory requests has kept the Resident Physicians on duty in the Laboratory so engaged that the clinical side of the cases was frequently lost to them. With the proposed changes, both the laboratory and clinical aspects of the ward cases will be kept in view, thereby making this department of the Hospital of still greater value to the clinician and the patient.

To complete our laboratory facilities, a thoroughly modern autopsy room and morgue should be provided, and, in connection with these, a suitable museum should be added. We are now in possession of valuable mate-

rial, both microscopic and gross specimens, which should be catalogued and arranged, that they would be readily accessible to study.

To the Board of Managers of the Hospital, who have always generously provided for the Laboratory, and to the Superintendent and Visiting and Resident Staff of the Hospital, many thanks are due, for helping to make the work easier, agreeable and profitable.

C. Y. WHITE,

*Director of Pathological Laboratory
and Curator.*

SUMMARY OF EXAMINATIONS.

Urine.....	7,776
Blood.....	3,236
Gastric analysis.....	114
Feces.....	110
Milk.....	342
Bacteriological cultures and examinations.....	3,409
Smears.....	2,204
Miscellaneous.....	324
Sputum.....	231
Autopsies.....	102
Histological preparations of autopsy tissue.....	1,431
Histological examinations of surgical tissue.....	1,832
Total.....	21,111

DETAILED EXAMINATIONS.

Urine.

Routine examinations.....	7,103
Special routine examinations.....	442
Albumen quantitative.....	36
Acetone.....	12
Bile.....	33
Blood.....	3
Cambridge test.....	4
Diacetic acid.....	8
Diazo.....	58
Indican.....	29
Sugar, special.....	20
Urea quantitative.....	3
Chlorides.....	14
Tubercle bacillus.....	9
Urobilin.....	6
Urorosinogen.....	1

Blood.

Differential leucocytic counts.....	410
Erythrocytic counts.....	338
General inspections.....	56
Granular degeneration.....	53
Leucocytic counts.....	1,902
Malaria inspection.....	139
Hemoglobin estimations.....	433
Coagulation test.....	6
Tubercular bacillus.....	1

Gastric Analysis.

Routine, chemical and microscopical.....	98
Occult blood.....	14

Feces.

Bile.....	4
Gall stones.....	1
Fat.....	9
Occult blood.....	62
Amœba.....	1
Parasites.....	20
Typhoid bacillus.....	20

Milk.

Bacteriological.....	171
Fat.....	171

Bacteriological Cultures and Examinations.

Blood cultures.....	97
For gonococcus.....	64
For diphtheria.....	127
Effusions.....	24
Miscellaneous smears from tissue and routine.....	2,200
Gauze.....	860
Water.....	1,072
Catgut.....	254
Air.....	212
Gloves.....	16
Drugs.....	23

Miscellaneous.

Cyto-diagnosis.....	35
Vaccines prepared.....	27
Microscopic examinations:—	
Dark field, blood.....	10
Wasserman reaction.....	284

Sputum.

For tubercle bacillus.....	220
For pneumococcus.....	11
Heart disease cells.....	5

General.

Autopsies.....	102
Pathological tissues from operations.....	550
Smeers, bacteriological.....	2,204
Histological examinations of surgical specimens.....	1,832
Histological preparations of autopsy specimens.....	1,431



FIG. 31.—Pathological Laboratory.



FIG. 32.—Pathological Laboratory.

REPORT OF THE X-RAY DEPARTMENT.

To the Board of Managers:—

The following is a list of the work done in the X-Ray Laboratory during 1912.

	Cases.	Plates.		Cases.	Plates.
Abscess:—			Bronchiactasis:—		
Diaphragm.....	1	1	Left.....	1	2
Ilium.....	1	1	Right.....	3	5
Jaw.....	3	4	Calcified glands in mesen-		
Abscess (tubercular):—			tery.....	1	3
Foot.....	1	1	Carcinoma:—		
Hip.....	2	2	Gall bladder.....	1	1
Sternum.....	1	1	Humerus.....	1	1
Adhesions posterior of ab-			Mouth.....	1	1
domen.....	1	1	Stomach.....	5	12
Aneurism.....	13	21	Uterus.....	1	1
Appendiceal sinus.....	1	1	Ventriculi.....	1	1
Ankylosis:—			Caries:—		
Elbow.....	2	3	Jaw.....	1	1
Arthritis:—			Stump of foot.....	1	1
Ankle.....	8	8	Cellulitis:—		
Elbow.....	9	10	Foot.....	1	1
Finger.....	1	1	Knee.....	1	1
Heel.....	1	1	Leg.....	3	4
Hip.....	7	9	Chronic gastritis.....	1	3
Hand.....	5	5	Chronic pleurisy.....	1	2
Knee.....	17	21	Chronic ulcerative phthisis...	1	2
Shoulder.....	9	9	Congenital dislocated hip....	3	3
Spine.....	1	3	Contusion:—		
Thumb.....	1	1	Ankle.....	26	27
Wrist.....	5	6	Arm.....	16	17
Foot.....	5	7	Back.....	10	11
Deformans:—			Chest.....	4	5
Hand.....	1	1	Elbow.....	32	36
Knee.....	2	2	Finger.....	11	11
Gonorrheal:—			Foot.....	18	19
Ankle.....	1	1	Forearm.....	5	5
Knee.....	2	2	Hand.....	12	12
Wrist.....	3	4	Head.....	1	1
Hypertrophic:—			Hip.....	7	8
Ankle.....	1	1	Jaw.....	4	4
Knee.....	2	2	Knee.....	15	17
Shoulder.....	1	1	Leg.....	11	12
Wrist.....	1	2	Pelvis.....	2	2
Peri:—			Rib.....	4	6
Shoulder.....	1	1	Sacrum.....	1	1
Rheumatoid:—			Shoulder.....	31	31
Ankle.....	2	2	Sternum.....	1	1
Knee.....	4	4	Thumb.....	3	3
Wrist.....	1	1	Wrist.....	7	7
Traumatic:—			Coxalgia.....	11	16
Knee.....	1	2	Cretinism.....	1	2
Shoulder.....	1	1	Deformity:—		
Thumb.....	1	1	Foot.....	1	2
Tubercular:—			Hand.....	1	1
Ankle.....	6	6	Duodenal ulcer.....	1	3
Knee.....	2	2	Empyema.....	7	9
Wrist.....	2	4	Endocarditis.....	2	2
Ascites.....	1	3	Enlarged bronchial glands...	1	2
Bladder calculus.....	18	40	Enlarged lymphatic glands...	1	2
Bone cyst of humerus.....	1	1	Enlarged mediastinal glands...	1	2

X-RAY DEPARTMENT—Continued.

	Cases.	Plates.		Cases.	Plates.
Epiphyseal separation:—			Foreign body (Con.):—		
Femur.....	7	7	Esophagus.....	9	12
Humerus.....	11	12	Shoulder.....	2	2
Radius.....	18	18	Spine.....	2	3
Tibia.....	5	6	Stomach.....	7	10
Ulna.....	1	1	Thigh.....	1	3
Exostosis:—			Trachea.....	4	6
Int. malleolus.....	1	1	Urethra.....	3	5
Os calcis.....	1	1	Wrist.....	3	4
Pubis.....	1	4	Gall stones.....	8	20
Ulna.....	1	1	Gastric ulcer.....	2	5
Extra dural hemorrhage.....	1	2	Hydrocephalus.....	1	2
Flat foot.....	4	7	Infection of finger.....	2	2
Fracture:—			Intercostal neuralgia.....	1	1
Accromial.....	3	3	Intestinal obstruction.....	1	2
Alveolar process.....	1	3	Knock knees.....	1	1
Astragalus.....	3	4			
Carpal.....	4	4	Luxation:—		
Clavicle.....	50	56	Ankle.....	1	2
Femur:—			Elbow.....	20	23
Neck.....	30	45	Finger.....	8	10
Shaft.....	40	51	Hip.....	5	6
Supra condyle.....	11	16	Hyothyroid.....	1	2
Fibula.....	58	70	Clavicle.....	3	5
Frontal bone.....	3	4	Knee.....	2	3
Humerus:—			Metatarsal.....	2	3
Deltoid bursitis.....	1	1	Semilunar cartilage.....	10	14
External condyle.....	15	20	Shoulder.....	14	17
Internal condyle.....	28	35	Spine.....	1	3
Intra capsular.....	5	6	Thumb.....	4	5
Shaft.....	41	64	Wrist.....	1	1
Surgical neck.....	26	41	Mitral regurgitation.....	1	1
Supra condyle.....	6	9	Mitral stenosis.....	1	1
Ilium.....	2	2	Myocarditis.....	3	6
Inferior maxilla.....	4	8			
Mandible.....	5	8	Necrosis:—		
Malar bone.....	2	4	Femur.....	5	9
Metacarpal.....	82	89	Humerus.....	1	1
Metatarsal.....	27	30	Jaw.....	3	4
Olecranon.....	19	21	Metatarsal.....	3	4
Os calcis.....	12	12	Ribs.....	2	2
Patella.....	23	32	Phalanges.....	1	1
Phalanges.....	36	38	Stump of phalanx.....	1	1
Pubis bone.....	1	1	Thumb.....	2	2
Radius.....	62	70	Ulna.....	1	2
Radius and ulna.....	218	244	Tubercular:—		
Ribs.....	23	25	Hip.....	1	1
Scaphoid.....	2	2	Knee.....	1	1
Scapula.....	6	6	Neuritis.....	2	2
Skull.....	4	6			
Tarsal.....	6	7	Osteomyelitis:—		
Tibia.....	38	49	Femur.....	3	5
Tibia and Fibula.....	60	85	Tibia.....	10	17
Ulna.....	21	25			
Vertebrae.....	2	3	Osteoperiostitis:—		
Foreign body:—			Clavicle.....	1	1
Abdomen.....	3	8	Fibula.....	2	3
Alimentary canal.....	2	4	Tibia.....	6	7
Back.....	1	1			
Breast.....	1	3	Osteophyte:—		
Buttock.....	1	1	Heels.....	1	1
Chest.....	3	6	Knee.....	1	1
Eye.....	20	37			
Finger.....	21	23	Ostitis:—		
Foot.....	8	10	Femur.....	1	1
Forearm.....	3	3	Knee.....	1	1
Hand.....	27	36	Paget's disease.....	1	2
Head.....	4	7	Periocarditis.....	2	2
Intestines.....	6	12			
Knee.....	3	5	Periostitis:—		
Leg.....	3	7	Fibula.....	1	2
Neck.....	1	1	Rib.....	2	2
			Tibia.....	5	6
			Pituitary body.....	1	1
			Pleurisy with effusion.....	1	1
			Pneumonia.....	5	6

X-RAY DEPARTMENT—Continued.

	Cases.	Plates.		Cases.	Plates.
Potts' disease:—			Sprain (Con.):—		
Cervical.....	1	1	Hand.....	6	7
Dorsal.....	4	4	Heel.....	1	1
Lumbar.....	7	10	Knee.....	10	12
Pulmonary tuberculosis.....	6	6	Shoulder.....	11	13
Pyelitis.....	1	3	Thumb.....	5	5
Redundant sigmoid.....	1	2	Wrist.....	56	58
Renal calculus:—			Synovitis:—		
Left.....	26	57	Hip.....	1	2
Right.....	38	90	Knee.....	9	10
Protrusion of tarsus.....	1	3	Shoulder.....	2	2
Rheumatism:—			Wrist.....	2	2
Wrist.....	1	1	Traumatic torticollis.....	1	1
Ruptured diaphragm.....	1	2	Tubercular:—		
Sacro iliac separation.....	5	8	Ankle.....	4	4
Sarcoma:—			Femur.....	3	3
Abdomen.....	2	4	Hip.....	8	10
Phalanges.....	1	1	Knee.....	1	1
Tibia.....	1	2	Pleurisy.....	1	2
Schlatter's disease.....	2	3	Spine.....	1	2
Sciatica.....	2	2	Tarsus.....	1	2
Sinusitis.....	1	1	Tumor:—		
Spondylitis:—			Abdomen.....	2	4
Cervical.....	1	1	Hand.....	1	2
Sprain:—			Thigh.....	4	5
Ankle.....	42	56	Unerupted teeth.....	1	4
Back.....	2	3	Visceroptosis.....	23	56
Elbow.....	16	16			
Foot.....	5	5	Total.....	2,076	2,705

(The number of plates does not nearly represent the number of exposures, as in many cases more than one exposure is made on one plate.)

Malignant Disease Cases, 48; Treatments, 518.

THOMAS S. STEWART, M.D.



FIG. 33.—Reception Room in the Nurses' Home.

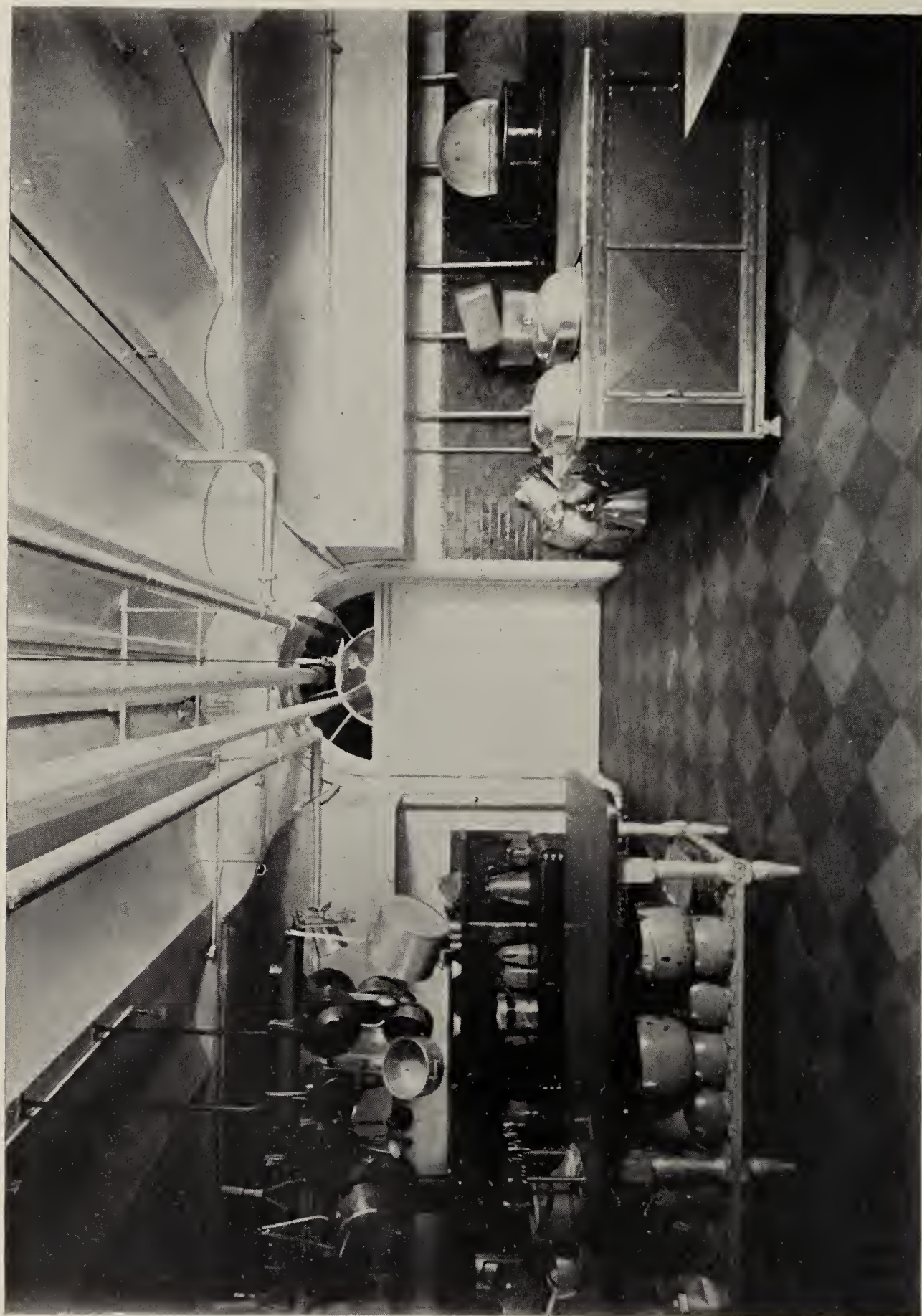


FIG. 34.—Central Diet Kitchen in Administration Building.

THE SURGICAL CLINIC OF THE PROTESTANT EPISCOPAL HOSPITAL OF PHILADELPHIA¹

REVIEW OF ONE HUNDRED AND FIFTY CONSECUTIVE
OPERATIONS

BY CHARLES HARRISON FRAZIER, M.D.
SURGEON TO THE HOSPITAL

THE series of cases herein discussed represent the surgical experience at the Episcopal Hospital in a three months' service, excluding a not inconsiderable number operated upon by one of the assistant surgeons or internes.

MORBIDITY. Of the 150 operations of our series almost 100 were for lesions of the abdominal organs. Of the total number there were four deaths, a mortality of about 2.5 per cent. Two of these four deaths, as will be seen, may properly be excluded, so that the mortality may be reckoned as 1.3 per cent. The series include 30 operations for appendicitis and its complications, 22 for hernia, 8 for lesions of the stomach or duodenum, 18 for operations on the pelvic organs with 4 hysterectomies, 4 operations on the biliary passages, 14 upon the urinary organs, and 3 thyroidectomies.

Of the four fatal cases one was a case of tuberculous meningitis, which, because of certain focal phenomena, was regarded before operation as a brain abscess. An exploratory craniotomy seemed justifiable. The patient survived the operation but a short time, and the autopsy revealed the true nature of the lesion. The second was a strangulated umbilical hernia

¹ Read before the Philadelphia Academy of Surgery, May 1, 1911. Reprinted from *Annals of Surgery*, October, 1911.

in an aged patient almost moribund, who scarcely survived the initial incision. These two might be regarded as inoperable.

The third death followed a suprapubic prostatectomy and bilateral herniorrhaphy under spinal anesthesia in a patient, aged seventy-five years. The patient's condition at the end of the operation was excellent and continued so beyond the period at which postoperative shock would have developed. But his vital processes gradually failed, and he died three and a half days after the operation. The fourth of the fatal series was in a patient who had been operated upon for gallstones. This case presented some interesting features and will be alluded to later.

TECHNIQUE. In the preparation of the field of operation the Grossich or iodine method was used, preceded by a single preparation with soap, alcohol, and bichloride. The addition of iodine to the technique has so diminished instances of accidental infection that I am disposed to omit altogether any attempt at chemical disinfection with alcohol and bichloride. There was but one frank suppuration in our wounds, and in that instance there were a few small pustules at the site of the wound before the operation to which the infection may be attributed.

In the postoperative period, after operations with incipient or advanced peritonitis, after operations in the upper abdomen, and in all cases of advanced years, with few exceptions, the patients were put in the sitting posture for at least forty-eight hours. This, I believe, minimizes the incidence of pulmonary congestion and in many instances adds materially to the comfort of the patient.

Proctoclysis was employed wherever for any reason the administration of water or liquids of any character by mouth was prohibited.

ANESTHESIA. The selection of an anesthetic deserves much more attention than it receives at the hands of most surgeons. No matter how skilled the anesthetizer may be in the administration of ether, there are certain cases where

for the comfort and safety of the patient either spinal or nitrous oxide anesthesia should be employed. While I do not believe nitrous oxide anesthesia will ever come to be universally used as a substitute for ether, there are many occasions where it is an important factor in the saving of life. In cases of severe toxemia, where patients are profoundly septic, as in some cases of gangrenous appendicitis, of peritonitis or gangrenous cholecystitis, in empyemas while resecting the rib, in suprapubic cystotomy for drainage or stone, in kidney decapsulation or nephrectomies in septic cases, or in strangulated hernias; in all these, as in many other minor procedures, there can be no question as to the value of nitrous oxide anesthesia. Its safety in competent hands is unquestioned; it relieves the patient of the ether discomforts, but above all it minimizes the risk of operation where life is hanging in the balance and the depressing effects of ether would be enough to turn the scale. Where abdominal relaxation is necessary, ether is substituted for a few moments until relaxation is obtained, returning to nitrous oxide.

Spinal anesthesia, too, has its special indications, it seems to me, particularly in patients with pulmonary tuberculosis and in elderly people, where, because of valvular lesions or myocarditis, the administration of nitrous oxide may be not unattended with risk. In this series I used spinal anesthesia in the following operations: A colostomy for inoperable carcinoma of the rectum; suprapubic drainage of a large abscess of the pelvic region in a septic patient; in a laparotomy for tubercular peritonitis and another for cirrhosis of the liver; in a suprapubic cystotomy; in a suprapubic prostatectomy; in the repair of a vesicorectal fistula. Scopolamine, gr. $\frac{1}{150}$, two hours before and morphine sulphate, gr. $\frac{1}{6}$, one-half hour before the operation so benumbs the sensibility of the patient, that, though conscious during the operation, he suffers from none of the ill effects which might come from fright in a wide-awake patient. From spinal and nitrous oxide anesthesia there were no ill effects. The only serious

complication occurred in an operation for hydrocele. The patient was an alcoholic; after a considerable quantity of ether had been administered without avail, chloroform was substituted, and just as the incision was made the heart stopped beating. Cardiac massage was resorted to by the transdiaphragmatic method, and in the course of a few moments feeble fibrillary contractions could be felt. Gradually the cardiac contractions became more perceptible and regular, and after ten minutes resuscitation was established. The abdominal wound was closed, and the operation on the hydrocele completed. The patient suffered no relapse, and the subsequent course of events was uneventful.

TUMORS. Curiously enough the district upon which the hospital draws for its material is not a fertile one for tumors. Relatively speaking there are few operations for cancer, exclusive of the uterus, and I have often attributed the paucity of malignant cases in the clinic to the popular impression among the less enlightened classes, that cancer is a disease of the blood and when removed by operation always returns in some other place. Of the benign tumors there were three unusual enough to deserve mention. One was a lipoma in the belly of the sartorius muscle, about eight inches below Poupart's ligament (Fig. 35). The tumor protruded above the surface only when the patient contracted the muscle. On first examination the character of the lesion was not suspected; it was thought to be a muscle hernia. The second was a tumor of the breast, which, because of the associated enlargement of the axillary lymph nodes, was thought to be either carcinoma or chronic cystic mastitis; but upon histological examination proved to be a fibroma. In the third case there was a large congenital angioma involving the palmar surface of the hand (Fig. 36) with digital extensions.

APPENDECTOMIES. In the management of the appendix cases no hard or fast rules were laid down. It was not our practice to attempt to classify our cases as early or late to



FIG. 35.—Photograph showing a fibrolipoma of the sartorius muscle mistaken before operation for a hernia. Note protrusion on the anterior surface of the upper portion of thigh.



FIG. 36.—Photograph of a congenital cavernous angioma of the hand with digital extensions.

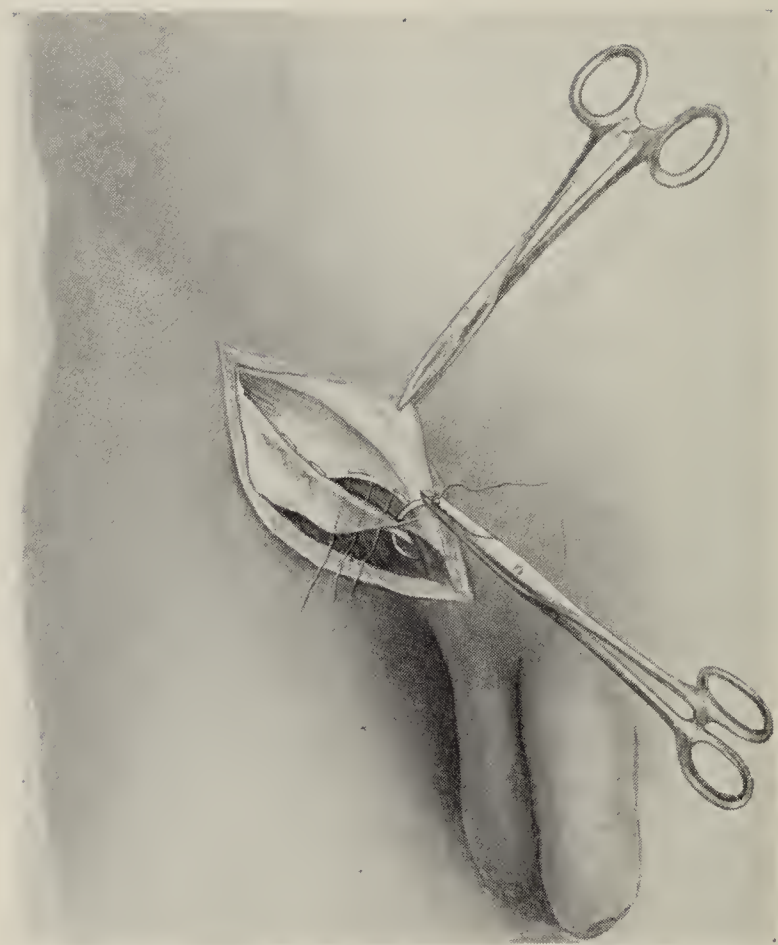


FIG. 37.—Application of the sutures in the operation for radical cure of inguinal hernia.

gratify the statistician, according to whether they were operated upon in the first forty-eight or seventy-two hours or later. In the acute cases with few exceptions the operations were performed as soon as the diagnosis was made. To this general rule there were certain exceptions. When the patient was first seen at the end or near the end of a mild attack we consulted our convenience as to the time of operation. Furthermore we advocated the starvation plan in a few cases of the following type: usually some time has elapsed since the onset of the attack; it may be only two days, more likely, however, three to five; the patient looks toxic, he is toxic; his pulse is rapid, over 120; the abdomen is distended and more or less tender everywhere; peristalsis has been altogether or partially arrested, perhaps reversed, with vomiting; restlessness and anxiety are written on the patient's face. In other words the infection is a widespread one. Sometimes this picture develops earlier, sometimes later, but whenever it comes I am convinced that the best results will be obtained by conservative practice, *i. e.*, by privation as to solids or liquids by mouth, proctoclysis, and posture.

One often hears or reads of bitter disputes as to whether the time limit between the early and late cases should be placed at forty-eight hours or later. Such discussions are futile, for early or late are but relative terms when applied to a given case. Each case must be a law unto itself, and no rules can be laid down comprehensive enough to allow for the innumerable variations.

It has been said, and very properly, that appendicitis is a surgical disease; no one denies it. The surgeon reserves as his privilege the right to decide upon the operability of a given case; that privilege is usually accorded him. In the exercise of this privilege he selects some cases for the Ochsner treatment and some of those cases may—before being operated upon—die. In the publication of his mortality statistics the surgeon usually excludes these. I believe, however, the time has come when the only means of determining the mor-

bidity of appendicitis is to include in our death rate those treated by both conservative and operative measures. We used to throw the responsibility on the medical men for all unoperated cases. But we cannot consistently do this any longer, if we take the stand, as I believe we should, that appendicitis is a surgical affection first, last, and always, and reserve the right ourselves to set aside a certain group, as I also believe we should, for conservative treatment. Having assumed entire responsibility for all cases, we must include in our statistics the fatal results in the unoperated as well as operated cases.

Irrespective of the appendices removed incidentally at other operations, there were thirty removed which were found at operation or in the laboratory to be diseased. There were no fatalities in this series, but *apropos* of what has just been said of the mortality statistics I should refer to the case of a young man, who when first seen on the fifth day of the disease, was desperately sick. I declined to operate, and watched him carefully from day to day. Under the treatment prescribed his improvement with each twelve hours was marked, until four days later the distention had subsided to the right iliac fossa, the pulse fell to 70, and the clinical picture was transformed from one of general to one of local peritonitis. I had planned to operate on the following day, but in the course of the early morning hours, the patient without warning developed signs of collapse, from which he did not rally. No autopsy was performed and the cause of death was a mystery. At the same time it should occupy just as conspicuous a place in my statistics as a death following operation.

The technique of the operation calls for little if any comment save with reference to the matter of drainage. In this I find myself using drainage in an increasingly smaller number of cases, and when drainage is required using less drainage material, rarely anything but split rubber tubing with a wick of gauze. The fundamental principle in draining

the peritoneal cavity is the relief of pressure afforded by the opening in the abdominal wall. This need not be larger than to accommodate one or two full-sized drainage tubes. I no longer leave a wound entirely open and filled with gauze, and I close wounds without drainage, sometimes even when there is a small accumulation around the appendix, thoroughly disinfecting the region with alcohol.

HERNIA. Of the hernia series there were 22 cases, of which 13 were indirect and 3 direct inguinal, 2 recurrent, 1 femoral, 2 ventral, and 1 umbilical. There were no unusual types except an irreducible omental hernia in a young adult, a case in which the diagnosis was not established until the operation. Without impulse it presented the earmarks of a lipoma. Of the series there was one fatal case in a large strangulated umbilical hernia of four or five days' duration, in the person of an aged woman who was generously referred to my service by a practising surgeon of a neighboring hospital. The patient was almost moribund at the time, and did not more than survive the hasty attempt to expose and free the contents of the sac.

The method of procedure in the inguinal type varied according to the character of the hernia; in some instances the simple Ferguson, in some the Bassini, and in others the imbricating method of Andrews was used. In the selection of the method I have always tried to observe the general principle of disturbing in the least degree the normal anatomical relations. Thus in the hernias of children and the incipient hernias of young adults where the rings are not large and the musculature all that could be desired it is not necessary to transplant the cord. After isolating the sac and freeing the neck from its attachments and ligating it an inch beyond the margins of the internal ring, the internal oblique and conjoined tendon were sutured to Poupart's ligament without transplanting the cord; and the wound in the external oblique sutured without imbrication. In complete hernias of longer duration with larger rings the

Bassini method was adopted. The remaining group included hernias of still longer duration, where the rings are more or less approximated, all direct hernias, all hernias in elderly people with musculature flabby and without tone, and hernias in which the conjoined tendon is altogether obliterated. Here I invariably resorted to the imbricating method of Andrews, in some cases splitting the sheath of the rectus and using either the belly of the muscle or flap of the sheath to fortify the defective area at the lower portion of the canal. This operation in this particular group is vastly superior to the Bassini method and should always be given preference. In all the operations, the incision in the external oblique is made a little above, instead of directly over, the canal and carried down, as Judd recommends above, and to the inner side of the internal pillar of the external ring so as to leave this structure intact. In introducing the deep mattress sutures in the reconstructive stage, my practice differs somewhat from the conventional procedure, in that I introduce the needle from the external aspect of the shelving edge of Poupart's ligament, and the knot is tied in the outer rather than the inner aspect (Fig. 37). This is a matter of but minor consideration, but has been adopted because it avoids the splitting or tearing of the edge of Poupart's ligament, which sometimes happens when traction on the suture is made to approximate the edges. For this suture I prefer to use a double strand of No. 0 or No. 1 catgut to a heavier single strand, as I always feel a little more confident of the sterility of the finer material.

ABDOMEN. We are gradually being weaned from the tradition that gallstones are innocuous in many instances throughout life to be discovered only at autopsy. That many cases of cholelithiasis have been treated for many years for stomach trouble we know too well, but in exceptional instances one runs across a patient in whose history there is nothing either indicative or even suggestive of the existence of gallstones.

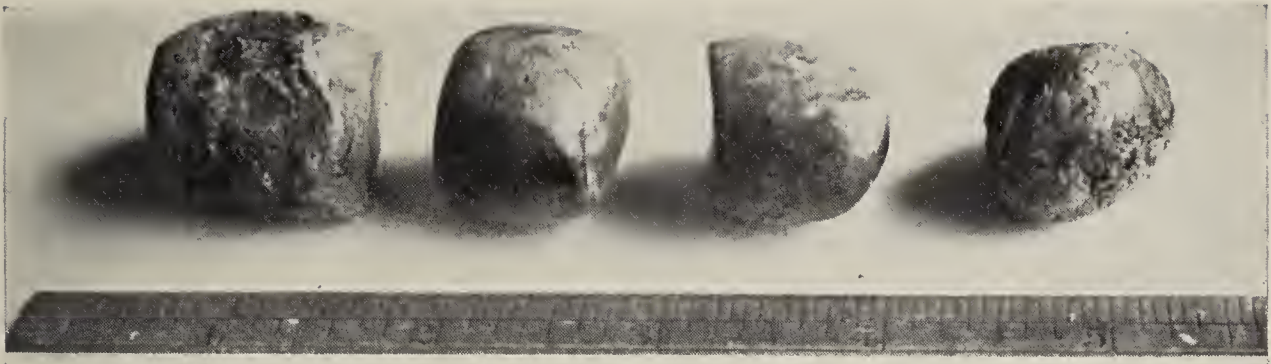


FIG. 38.—Illustration showing series of gallstones arranged in column as they were found imbedded in a mass of inflammatory tissue, after having escaped from the gall-bladder, of a patient who had been free from symptoms until within two months of the date of the operation.



FIG. 39.—Portion of stomach removed, showing in centre of specimen the cavity of an indurated ulcer on the lesser curvature.

Such was the case in K. S., aged fifty years, who told me she had never been sick in bed, that she had never been treated for dyspepsia, and had none of the digestive disturbances so common in cholelithiasis, much less any of the acute exacerbations of biliary colic or subacute cholecystitis, at least until two months before the operation, when she had attacks of pain in the upper abdomen, which had confined her to bed for a while. I questioned her myself with great care, as the physical signs pointed to the biliary passages. There was a mass in the abdomen about the size of a fetal head, extending from the margin of the ribs to the umbilicus. It did not move freely with respiration, and was not continuous with the margin of the liver. Through a right rectus incision I came down at once upon a mass, surrounded by adhesions, which were separated with great difficulty. Finally a cavity was opened, containing pus and four large gallstones (see Fig. 38). These were firmly imbedded in the inflammatory mass, arranged in single column, and faceted at either end, where they were in direct contact. There was no escape of bile at any time, and nothing which could be identified as a gall-bladder was seen. The gallstones, which must have been there months, if not years, had ulcerated through the gall-bladder and became imbedded in a mass of inflammatory tissue. For two days the patient's condition was most satisfactory, but on the third day there was for the first time a copious discharge from the wound, which was believed to be bile. On the fourth day 90 ounces of this fluid were collected.

Her pulse was growing more rapid and weaker, her skin leaky. I was at a loss to account for her rapidly failing strength. I began to suspect the fluid, draining in such quantities, was not pure bile; a specimen examined disclosed hydrochloric acid, and further investigation pointed to a fistulous communication with the stomach and duodenum, probably resulting from the trauma incidental to the liberation of the gallstones from the inflammatory bed. The patient was taken to the operating room again, and through a left rectus incision our suspicions were confirmed. The tissues about the perforation were so fragile that it could not be satisfactorily closed with sutures. Accordingly a gastrojejunostomy was performed and the pyloric outlet closed. The patient did not react and died the following day. Had the true nature of this lesion been recognized sooner, I cannot but feel she would have survived the second operation.

Of the lesions of the upper digestive tract there were in all 9 cases; 2 gastric carcinomas, 2 gastrophtoses, 3 gastric and 2 duodenal ulcers. All were operated upon and there were no fatalities. It is interesting to note that in the ulcer cases the proportion of males to females was 4 to 1, a further evidence of the greater prevalence of ulcer in the male sex.

Before discussing the operative procedures in this series I will refer to some interesting facts bearing upon the diagnosis and symptomatology.

In one case, B. F., an engineer, aged fifty-five years, the pre-operative diagnosis was cholelithiasis. There was a history of stomach trouble covering a period of two years, with bilious attacks and occasional vomiting; the patient stated that he had been jaundiced off and on, that his stools had been clay colored, and that discomfort followed eating. There was some rigidity and tenderness over the gall-bladder, and the scleræ were jaundiced. At the operation a typical saddle-back ulcer was discovered, with an unusually extensive infiltration of the gastro-hepatic omentum. To the encroachment of this upon the common duct we attributed the signs of obstructive jaundice, which led us astray in the diagnosis. The ulcer-bearing area with a portion of the gastro-hepatic omentum was resected, and all the symptoms, including those of common duct obstruction, disappeared (see Fig. 39).

The unreliability of the gastric analysis in the diagnosis of gastric or duodenal ulcer or cancer was forcibly illustrated in our cases. As to the presence of hyperacidity or of occult blood in the contents of the stomach or bowel, I am becoming more and more convinced that to wait for such laboratory indications of ulcer is unjustifiable in the presence of a reasonably clear history. It is after all the history upon which we must place the most reliance, and upon which our decision for or against operation must be founded. Unfortunately the "history," so-called, is often not an accurate record of the development of the disease in chronologic order, but a collection of isolated facts forced from the patient by a system

of cross-examination at the hands of one who often approaches the case with preconceived notions as to the diagnosis and conducts his examination accordingly. A better term, as some one has recently pointed out, is anamnesis, which implies a record of the disease from the patient's recollection, and if the patient is intelligent enough it should be written by the patient and not for him. That one may sometimes be misled, however, by the history is shown in my experience with a young woman evidently neurasthenic in temperament. Although skeptical about the existence of a gross lesion, I was finally persuaded to explore the upper abdomen, because of the very positive evidence of hematemesis and of aggravated attacks of vomiting and pain. The findings at the operation were negative and I ascertained afterward from the patient that she was in the habit of sucking the gums until they bled, swallowing the blood, and then inducing vomiting. Hence the hematemesis.

To me an interesting case, because of the difficulty in diagnosis and the duration of the disease, was that of a young man, aged thirty-one years, who had been ailing twenty years off and on; at the age of eleven he had an attack of vomiting with abdominal pain which confined him to bed for ten days; and even prior to that time had what he called indigestion. He had served in the army during the Spanish War, and since then had worked steadily as a machinist. His work had never been interrupted, he ate everything, had never vomited, and never had any blood in his stools. His chief and only complaint was pain, worse when the stomach was empty, often having to get up at night for a glass of hot milk or water. There was no occult blood, no hyperacidity, no dilatation of the stomach. He was tender over his epigastrium but he was tender also over his appendix. Although the clinical picture did not conform altogether to type, I thought we were dealing with a case of appendicular dyspepsia, a chronic appendix with gastric symptoms, and proceeded accordingly. Through a gridiron incision I explored the appendix, and found it buried in a mass of adhesions, and liberated it with much difficulty. Though there was no doubt as to the existence of a lesion of the appendix, I was still in doubt about the upper abdomen, which

I explored through a right rectus incision and found the stomach and omentum plastered to the parietal peritoneum, and on further investigation an ulcer of the lesser curvature. From the extent of the peritoneal invasion there had evidently been at one time some leakage. The operation concluded with a gastro-jejunal anastomosis. Recovery was uneventful.

The method of procedure in gastric ulcer necessarily varies. However, I strongly advocate excision of the ulcer when this is feasible, and especially in the large indurative type, including the saddle-back ulcer in patients approaching middle life. It is in this type that carcinoma is most frequently implanted, and for this reason alone, if for no other, we owe it to our patients to practise the more radical procedure. These ulcers should be looked upon as precancerous conditions and treated accordingly. When the ulcer is so situated or so adherent to adjacent structures as to make excision difficult, the pyloric outlet of the stomach should be closed by infolding as the surest safeguard against recurrence and as the most rational way (in the light of our present knowledge of the pathogenesis of ulcer) of securing permanent results. The same practice is indicated, I believe, in duodenal ulcer as well, although in my experience the tendency to recurrence in the latter is not as great as in gastric ulcer. The operations in this series of gastric cases were carried out according to these principles. In technique the no-loop gastro-jejunostomy with vertical opening was used, and the line of sutures protected, as Mr. Moynihan suggests, with the gastro-colic omentum.

The management of actively bleeding ulcers requires the exercise of one's discretion. Ordinarily I favor palliative procedures.

For example one of our patients, a young woman of twenty-two, with a five-year ulcer history, had three large hemorrhages in five days, once vomiting three quarts and with a large quantity of blood in the stools. On admission she looked exsanguinated; her hemoglobin was low. I decided not to operate at once, ordered

20 c.c. of horse serum given hypodermatically, morphine q.s. to allay pain and restlessness, and small quantities of saline solution by bowel, and an ice-bag to the abdomen. After five days of freedom from hemorrhage or vomiting I operated and found an ulcer on the greater curvature. If, on the other hand, the plan of treatment had been of no avail and hemorrhages recurred, with increasing frequency or in increasing amounts, there would then have been but one recourse, immediate operation.

Of the pelvic cases, in fact of the entire series, the most puzzling was in the person of a patient, aged fifty years, referred to me by Dr. E. F. Walsh. She told me that she had not been well since the birth of her last child three years ago, and prior to that had had one miscarriage, that her menstruation had been regular until the last period, now two weeks overdue. She laid emphasis on troublesome attacks of indigestion with vomiting, for which her physician had treated her from time to time. Eight days prior to admission, after a hearty meal, she was seized with a violent pain and vomited. For the next three days vomiting continued but the pain was less severe. She remained in bed with a normal temperature on a liquid diet, and on the day before admission she had had another attack of severe pain similar to the first. When I saw her she appeared exsanguinated, her hemoglobin was 20 per cent., the leukocytes 22,400, and pulse 140; the abdomen was distended, and she referred her pain to the epigastrium and precordium. My first thought in view of the history of digestive disturbance, the intense pain, and attacks of vomiting was hemorrhage from a duodenal ulcer. In her present condition operation seemed out of the question, so I watched her carefully day by day, and gradually the symptoms of the upper abdomen disappeared and were replaced by an area of dulness and tenderness in the lower abdomen. To make a long story short, when her condition justified it I opened the abdomen and found we were dealing with an ectopic gestation. From a pint to a quart of partially organized blood-clot was removed together with the sac. There was no post-operative shock, and convalescence was rapid and uneventful.

Unfortunately the Episcopal Hospital is so far removed from the centre of the city as to be inaccessible both for the casual visitor and student alike. This isolation is a great drawback, as there is nothing so stimulating to the whole staff of a hospital, from the highest to the lowest officer, as

the constant criticism and searching inquiry of those who are seeking useful information in modern methods of procedure. Because of its isolation the physical equipment of the hospital is known to few of the profession, resident and non-resident. Perhaps the most favorably situated of the larger hospitals for service to the working classes, it has a wealth of material representing the whole field of surgery. Of the lesions of the extremities, there is an extraordinary opportunity to study fractures of every description, and of the abdominal lesions there is a veritable mine of examples of inflammatory conditions of the pelvis, most of which are the result of improper care at childbirth at the hands of incompetent midwives or the patients themselves. To the philanthropically inclined I know of no better object for investment than the establishment of a maternity department for the poor of that district.

The service of this hospital, as of many others, is an interrupted one. The disadvantage of this system pertaining in many institutions is well recognized and so patent as to admit of no dispute. Far better is the European system, where in each hospital a limited number of surgeons devote their entire time to the instruction of young physicians and surgeons and the utilization of the material at their command for the advancement of medicine as a science and an art.



FIG. 40.—Carbolic acid gangrene. Case I.



FIG. 41.—Carbolic acid gangrene. Case II.

CARBOLIC ACID GANGRENE. REPORT OF TWO CASES¹

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CASE I.—Sam S., aged twenty-five years, mashed the end of his left index finger one afternoon about four o'clock. During the evening he consulted his family physician, who advised him to tie up the finger in rags soaked in a solution made by adding two drops of pure carbolic acid to a glassful of water. The advice was followed; the patient, and his sister, who helped him to tie his finger up, both assert that not more than two drops of the strong solution were used, and that this amount was diluted in a glassful of water. When the rags were removed the next day, the end of the finger was black and numb.

Four weeks later (October 10, 1912) the patient came to the Surgical Dispensary of this hospital, in Dr. Ashhurst's service. He had not returned to the physician whose advice he had sought at first, but had taken care of his finger at home. The condition when seen in the Dispensary is well shown in the accompanying photograph (Fig. 40); the finger was black, desiccated, and shrivelled, with a distinct line of separation.

Amputation at the proximal interphalangeal joint was advised, but refused. It was learned subsequently that the patient afterwards went to another hospital, and finding that the same advice was given, finally consented to have the gangrenous finger removed.

It seems scarcely probable that so weak a solution for so short a time, as was used by this patient, can have had so disastrous an effect. It is possible, of course, that he used

¹ Read before the Episcopal Hospital Clinical Society, November 18, 1912.

more than two drops of the strong carbolic acid solution, or that he diluted it with less than a whole glass of water, or that he kept the dressing on his finger longer than he acknowledged. There is little doubt that as evaporation takes place a solution originally weak becomes stronger, but at any rate this case merely serves to emphasize once more the very real danger that exists in the indiscriminate employment of carbolic acid as a dressing. The following case, seen some years ago in Dr. Ashhurst's service at the Episcopal Hospital, is even more surprising.

CASE II.—A child, aged three years, received a slight cut on the finger, and her mother tied the wound up in rags soaked in "phenol-sodique" a popular and much advertised household remedy. When the rags were removed the next morning the skin of the finger was found to have turned black, and the mother brought the child to the Surgical Dispensary, January 15, 1911. There it was found that gangrene of the skin had occurred already (Fig. 41), but fortunately the deeper parts did not seem to be involved; and under simple dressings the skin sloughed off, leaving a healthy granulating surface. Though healing was slow, it was eventually complete, a very useful finger being retained.

This case well exemplifies the value of the rule that in cases of carbolic acid gangrene amputation should be delayed until the extent of the damage is clearly defined. In the first case reported, complete gangrene of the finger up to the middle of the second phalanx had taken place before the patient came under observation, and there was nothing to be gained by delay. In the second case a prudent delay gained for the child a very useful finger.

THE ANTITOXIN TREATMENT OF TETANUS¹

BY ELLIS E. W. GIVEN, M.D.
SURGEON TO THE DISPENSARY OF THE HOSPITAL

THE treatment of tetanus by means of its specific antitoxin is sharply divided into two distinct phases, one dealing with the prevention of the disease, the other with the treatment after symptoms have developed.

Shortly after the discovery of the antitoxin for this disease it was believed all that was necessary to cure the disease after symptoms had developed was to inject sufficient antitoxin to neutralize the amount of toxin then present in the body, but that this was erroneous was proved by the high death rate following such serum treatment.

Tetanus toxin combines with nerve tissue, and when so combined antitoxin is powerless to break up the union, but is capable of neutralizing all toxin in the body not so combined if injected in sufficient dose and brought in direct contact with it.

If a quantity of tetanus toxin known to be fatal to animals is mixed with sufficient antitoxin, the mixture thus made can be injected into the animal without fear of tetanus resulting, the antitoxin having combined with the toxin forming a nontoxic mixture.

Toxin and antitoxin when injected simultaneously but at different points are absorbed with different degrees of rapidity. The toxin is absorbed more rapidly than the antitoxin and tetanus will develop to a mild but curable degree.

¹ Read before the Episcopal Hospital Clinical Society, November 18, 1912.

According to Vaillard, injections of serum made during the incubation of the disease will give results varying according to the dose given and the tissue into which the injection is made. If the injections are made into the cellular tissue the preservation of the animal is almost certain, while if the virus is injected into the muscle without being neutralized with the antitoxin, the antitoxin being given elsewhere into the body, it is doubtful if the animal can be saved.

When an individual receives a wound contaminated with street dirt, garden earth, stable manure, or other tetanus-bacilli-bearing matter, living bacilli are thus injected directly into the body, and if there is broken down tissue, blood-clot, or a suppurating wound, they begin to grow, and, after a time varying from a few days up to eight, ten, or more days, symptoms of tetanus develop, the toxin liberated from the growing bacilli having combined with the nerve tissue.

Antitoxin injected about the same time that the wound occurred would be in the body ready to neutralize the toxin as liberated from the growing bacilli before it combined with the nerve tissue, for after combinations with nervous tissue antitoxin cannot break up the combination thus formed.

Dehne and Hamburger have carried out experiments to determine the duration of immunity in man, and report that the blood in the injected man rapidly increases in protective power up to the second or third day after injection, to remain constant to about the seventh or eighth day, after which time a rapid fall in protective power is noted and only slight trace can be found up to the twentieth day.

In animals, especially the horse, injected for prophylactic purposes, the immunity to tetanus lasts much longer than it does in man, and this lengthened time of immunity is explained by the fact that the horse serum which is the usual medium for conveying the antitoxin to man is, when injected into the human being, a foreign protein and as such is rapidly expelled, but when injected into the horse it is a normal constituent of that animal's body and is retained for a much

longer period of time, conferring immunity for the full period.

According to Nocard and Leclainche the prevention of tetanus following traumatism is assured by injection of tetanus antitoxin; their belief being founded on results following the prophylactic injection of over 3000 animals.

In the early days of the manufacture of antitoxin there was no fixed standard by which to judge of the potency of the product used and results differed widely according to the serum used. Prior to 1908 this condition held throughout the world, but from this date (1908) manufacturers began to standardize their products. France at present gives a protective dose of about 500 units; Germany 2000 units, while the standard prophylactic dose in the United States is 1500 units.

Antitoxin, being able to combine only with the toxin that is free in the blood and lymph channels, is able to act only so long as the toxin is thus contained; it is therefore necessary to begin prophylactic treatment as soon as possible after the receipt of a traumatism suspected or known to carry tetanus infection. As soon as possible after such a wound is received it should have a full, free, and open cleaning, a general anesthetic being administered whenever the condition of the wound or patient renders local anesthesia undesirable.

Incisions should be such that will best expose the wounded areas, both superficial and deep, all foreign material, blood clots, torn and lacerated or severely bruised tissues, must be removed and the wound made to be as clean and free as possible of everything which would cause or tend to cause subsequent breaking down of the surrounding tissues, or suppuration within the wound. Manipulations must be made gently and with light touch, no caustics or irritating chemical solutions or acids are to be used. After all parts of the wound, including obscure pockets, etc., have been thoroughly but gently treated, at least 1500 units of a recent and standard tetanus antitoxin must be injected into the patient.

This may be injected into any part of the body, but is preferably made into or near the original wound, so that the nerves, injured by the accident and near to the site where the probably introduced tetanus bacilli will grow and give off their toxin for absorption, will most readily take up the newly formed toxin. The wound is to be left as an "open wound," lightly packed with 10 per cent. iodoform gauze or plain gauze followed by sterile gauze dressing and bandage. Redressing should take place at least once every twenty-four hours, when all parts of the wound are to be exposed to the air and again dressed as on the first day.

If the wound is a severe one with prolonged suppuration favoring the retention and growth of living tetanus bacilli, or there is discharge of broken-down tissue from the wound, then in addition to the daily dressing subsequent injections of tetanus antitoxin should be given every seventh or eighth day, dating from the day of first injection of antitoxin, so long as suppuration persists, for, as has been cited, the blood-contents of antitoxin become almost absent after the end of the first week and toxin liberated from the retained tetanus bacilli present in the prolonged suppurating wound would, after the end of the first week, find no antitoxin present to combine with and neutralize it. In this way are explained those cases of late development of tetanus in patients with prolonged suppurating wounds who, shortly after the receipt of the injury, had received one prophylactic dose of antitoxin but none subsequently.

Should the wound be such that bony tissue is attacked and necrosis occurs this must be removed fully and freely, for tetanus is likely to develop in a wound once infected with tetanus bacilli if suppuration continues.

The healing of the wound and the prevention of tetanus depend, as Vaillard says, upon three agents:

1. Antitoxin, to neutralize the toxin present.
2. Phagocytosis, to deal with the bacilli in absence of the toxin.

3. The surgeon, who shall with care remove all blood-clots, foreign material, bruised or broken-down tissue, and leave the wound in a condition for healing.

When treating this severe and dangerous disease no effort should be spared to obtain and use only the most reliable material and preparations; dried serum containing the antitoxin is not recommended. If this is dusted into the wound absorption may take place, but dependence cannot be placed on it. The only time dry serum is permissible is when it is the only serum obtainable; it may then be used but never is it to supersede the standard liquid serum of known antitoxin content.

For a number of years the writer has made it a practice to administer a prophylactic dose of tetanus antitoxin to all patients having wounds contaminated with street dirt, garden earth, etc., entering his service at the Episcopal Hospital, or seen in private practice, and none of these patients thus injected, so far as he knows, has developed tetanus. Recently a collection and study has been made of all cases of tetanus admitted to the Episcopal Hospital from 1905 to 1912, and none of these cases had had a prophylactic dose of tetanus antitoxin.

The treatment of the disease after symptoms have set in has to deal with a condition where there is an enormous amount of highly potent toxin circulating freely in the body and great quantities are rapidly being formed and absorbed, while an unknown amount has already been absorbed into the nervous tissue.

Treatment should be begun at the earliest possible moment by eliminating from the body as much of the toxin as possible, by free venesection and lumbar puncture, and the injection of huge doses of antitoxin by subcutaneous, intravenous and intraspinal methods to neutralize the unabsorbed toxin circulating in the body, together with intraneural injections to reach and neutralize the toxin contained within the nerves. No limit should be placed on the amount of antitoxin to be

injected, if obtainable, for it will surely neutralize all the unabsorbed toxin and if this is neutralized and the bacilli have been removed by the surgeon so that no more toxin is formed to be thrown into the system for absorption, the body may be able to combat successfully the amount already combined with nervous tissue. If antitoxin is withheld there is nothing to neutralize the great quantity of uncombined toxin which then will soon combine with nervous tissue and add its toxic power to the unknown amount already combined.

The local treatment of the wound, if it can be found, must follow much the same lines as noted for the prophylactic treatment. A general anesthetic (often indicated to control the spasms) must be given in order thoroughly to clean out the wound. If it has closed it must be freely opened, importance being placed on local cleansing of the wound, for the tetanus bacilli are always a local infection, rarely being found away from the original site of injury. All blood-clots, broken-down or suppurating material must be carefully removed with light and dexterous manipulations, great care being exercised to produce no bruising of the tissues which might give resting place for growth of any bacilli unintentionally left in the wound.

After thorough cleaning the wound is to be lightly packed with iodoform gauze or plain gauze, as noted under prophylaxis, which dressing is to be repeated at least once every twenty-four hours.

Supporting systemic treatment is indicated, together with highly nourishing food in liquid form given, if necessary, through a stomach tube, and sedative drugs are to be administered as indicated, the patient being treated in a quiet and darkened room.

BIBLIOGRAPHY

- Behring and Kitasato. Deut. med. Woch., 1890, xvi, 1113 to 1114.
- Reck. Zeit. für Veterinär., Jahrg. 13, May, 1901, Heft v, p. 214.
- Hutyra and Marek. Spezielle Pathologie und Therapie der Haustiere, Jena, 1909, vol. ii, Zweite auflage.
- Richmann. Berlin. tier, Woch., November 4, 1909, xxv, No. 44, 801 to 802.
- Nocard and Leclainche. Les Maladies Microbiennes des Animaux, 1903, vol. ii, third edition, Paris.
- Vaillard. Sur les injections preventives de serum antitoxique dans e prophylaxie du tétanos de l'homme. Bull. de l'Acad. de Méd., May 26, 1908, pp. 567 to 577; June 2, 1908, pp. 584 to 597.
- Labat. Sur l'emploi du serum antitétanique. Rev. Vét., September 1, 1902, xxvii (lix), 580 to 586.
- Naudrin. Ann. de Méd. Vét., August, 1903, Tome lii, No. 8, pp. 442 to 446.
- Huguier. Rec. de Méd. Vét., June 15, 1909, Tome lxxxvi, No. 11, pp. 357 to 360.
- Dieudonné. Rec. de Méd. Vét., July 15, 1909, Tome lxxxvi, No. 13, pp. 433 to 437.
- Labat. Rev. Vét., September 1, 1909, Tome xxxiv (lxvi), No. 9, pp. 537 to 542.
- Pecus. Jour. de Méd. Vét. et de Zoötech, October 31, 1909, Tome lx, pp. 591 to 593.
- Rapport de Commission. Rec. de Méd. Vét., March 30, 1910, Tome lxxxvii, No. 6, pp. 125 to 141.
- Hitchens. Amer. Vet. Rev., August, 1910.
- Pearson. Amer. Vet. Rev., September, 1897, xxi, No. 6, 380 to 386.
- MacFarland and Ranck. Jour. of Comp. Med. and Vet. Arch., vol. xx, Nos. 10 and 11, pp. 639 to 646.
- Nocard. Amer. Vet. Rev., October, 1897, pp. 453 to 462.
- Dehne and Hamburger. Wien. klin. Woch., July 4, 1907, Jahr. vol. xx, No. 27, pp. 817 to 823.
- Römer und Sames. Zeit. für Immun., December 28, 1909, Orig. Band iv, No. 3, pp. 270 to 285.
- Rosenau and Anderson. Bul. No. 43, Hyg. Lab., United States Public Health and Marine Hospital Service, Washington, D. C., March, 1908, pp. 1 to 55.

THE RATIONAL TREATMENT OF TETANUS, WITH
A REPORT OF TWENTY-THREE CASES
FROM THE EPISCOPAL HOSPITAL
PHILADELPHIA¹

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THIS paper is written with three purposes: (1) To report 23 consecutive cases of tetanus treated at the Episcopal Hospital, Philadelphia, during the last eight years; (2) by a review of the recent literature to endeavor to inculcate rational methods of treatment; and (3) in the hope that others may profit by our mistakes, as we have profited ourselves. For permission to record these case histories, as well as for the privilege of having under our personal care 11 of the patients, we desire to express our cordial thanks to the various members of the staff in whose services the patients were treated.

It is necessary first of all to inquire whether there is any such thing as a rational treatment for tetanus. We hold that treatment can be rational only when it is founded on a knowledge of the pathogenesis of the disease in question; and we believe that in the case of tetanus such knowledge exists.

Though previous generations had enough clinical acumen to class tetanus among diseases of the nervous system, and

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though the discovery of the *Bacillus tetani* by Nicolaier in 1884, and its isolation in pure culture by Kitasato in 1889, proved it to be an infectious disease, yet it is only within the past ten years that any definite knowledge as to its pathogenesis has been acquired. This knowledge has been secured almost solely through animal experimentation, and it is only as a result of the information gained in this way that rational treatment has become possible. Previous treatment, even if successful, was purely empirical. Especially noteworthy in the development of our knowledge have been the elaborate researches of Marie and Morax (1902), Meyer and Ransom (1903), Sawamura (1909), and v. Graff (1912). We have drawn freely from the various publications of these authors, as well as from Frazier's excellent critical review of the subject.

It is known that the disease is a pure toxemia. The bacilli or their spores may exist indefinitely in the tissues, and *no symptoms will be produced unless toxins are formed*. If the toxin is introduced into the system, it produces all the characteristic symptoms of tetanus even though no bacilli are present.

EXPERIMENTAL TETANUS. *Tetanus Ascendens*. In the small animals used in laboratory work the symptoms of the disease almost always begin in the inoculated extremity (*local tetanus*), and though other neighboring parts may become affected subsequently, yet trismus and retraction of the head almost never are observed, the disease being terminated by death or recovery before this stage is reached. In such animals (rabbits, rats, mice, frogs) if one lower extremity is inoculated with tetanus toxin the symptoms develop first and are most pronounced in that extremity; the opposite lower extremity is involved later, but not to the same extent; and when the spinal muscles become affected those of the injured side frequently are more involved than those of the uninjured side, thus resulting in pleurothotonos. The disease in such cases is known as *Tetanus Ascendens*;

its symptoms develop first in the injured part and gradually ascend.

Tetanus Descendens. Usually in the larger animals and in man, in whom the disease is acquired not by the injection of toxin, but by inoculation with the bacilli of tetanus, the symptoms begin first in the muscles of the neck and jaws, *no matter where the point of inoculation*. Subsequently the muscles of the back and trunk are affected, and finally, those of the extremities, especially the lower extremities. The disease in these cases is distinguished as Tetanus Descendens. In man ascending tetanus is not extremely rare (Cases 2 and 4 of our series), though the initial symptoms (cramps and stiffness in the wounded extremity) often are overlooked, and the case is classed as one of pure descending tetanus. Mixed forms also exist, in which the disease seems to run both an ascending and a descending course.

Is there a satisfactory explanation for the difference between these two forms of tetanus, ascending and descending—the former being that which is produced experimentally, and the latter that which occurs in nature? Zupnik in 1900 found that he could produce descending tetanus experimentally, even in small animals, provided the toxin was injected into muscle-free parts of the body, such as the tips of the toes (rabbits). These results were confirmed in 1909 by Sawamura, and it was he who advanced the true explanation for the difference between tetanus experimentally produced and that which occurs in nature. In experimental animals the toxin always had been injected into the muscular parts (usually the thigh or calf of the leg), and here it came into relation almost immediately with the motor nerves, was absorbed rapidly by them, and the part of the spinal cord first to be affected was that in anatomical relation with the wounded limb. In man, however, the usual point of inoculation is the hand or foot, and while a certain amount (perhaps a large amount) of toxin eventually reaches the nearest motor nerves and ascends them to the spinal cord, yet other

portions of the toxin are taken up first by the lymphatics, then enter the general circulation, and passing out from the left heart are carried in short order to *the end-plates of all motor nerves throughout the body*. The portions of toxin reaching in this way the end-plates of such short nerves as those of the facial, cervical, and spinal muscles will reach the spinal cord through these short nerves, and will begin to produce tetanic spasm in the parts supplied by these short nerves even before the toxin in the wounded extremity has had time to ascend the nerves of that extremity as far as the anatomically related region of the spinal cord. In this way, therefore, descending tetanus is to be explained. And Sawamura found that among 23 reported cases of ascending tetanus in man the point of inoculation was in a muscular part in every case but one, thus strengthening his view that the occurrence of ascending tetanus is due to the ready access of the toxin to the motor nerves. To prove this supposition still farther, he showed experimentally that if the nerves of an extremity are divided no local (ascending) tetanus can be produced by the injection of toxin; and that even if descending tetanus finally develops after many times the usual lethal dose of toxin has been injected, yet no local tetanus occurs in the enervated extremity.

Cephalic tetanus, as seen in man following wounds of the head or face, is to be explained in this way: It is a local tetanus, and it occurs first in the wounded part, and is manifested first by facial spasms and paralysis, because the toxin finds ready access to the short cranial nerves and reaches the centres which control them long before the toxin admitted to the general circulation reaches the spinal cord through other, longer nerve trunks.

PATHOGENESIS OF TETANUS. 1. *The toxin ascends the peripheral nerves to the spinal cord*. From what has been said above it is evident that the symptoms of tetanus are due to the action of the toxin on the central nervous system, especially to its action on the spinal cord. It was shown by Marie

and Morax in 1902, by Meyer and Ransom in 1903, and has been repeatedly demonstrated by experimenters since their time, and it may be accepted as irrefutably proved, that in ascending tetanus the toxin ascends the nerves of the wounded part (especially the motor nerves) until it reaches the spinal cord; that only when it reaches the cord does it begin to produce symptoms; that the first symptoms produced are in the parts supplied by the portion of the cord first attacked; that the toxin spreads in the cord, up or down, more especially ascending it; and that it produces symptoms in the parts supplied by every portion of the cord attacked. It is known that tetanus toxin can and does exist free in other portions of the body, such as the internal organs (liver, spleen), where it may be found after death; but that only those portions of the toxin which reach the spinal cord are able to produce recognizable symptoms.¹ And it is known that after death little or no toxin can be found in the spinal cord. At first sight this seems paradoxical, but the explanation is that when the toxin reaches the cord it soon enters into combination with the nerve tissue, becomes impregnably entrenched there, and cannot be dislodged by any known methods.

The peripheral nerves have little power of neutralizing the toxin—no more power than has the liver, for instance; and after death toxin may be recovered from the nerves as readily as from the internal organs. The peripheral portions of the nerves are found to give up more toxin than those portions nearer the spinal cord. In other words, *the nearer the toxin gets to the spinal cord the more impregnably entrenched does it become in the nerve tissue*. A much larger amount of toxin must be injected into the periphery of the limbs (*e. g.*, subcutaneously in the toes) to cause death, than in the central portions of the limbs (*e. g.*, intramuscularly);

¹ In animals rapid emaciation sometimes has followed administration of tetanus toxin, when no tetanic symptoms were produced. Probably these cases, which are described as *tetanus sine tetano*, are due to the action of the toxin on structures other than the nervous system.

in other words, *the readier the access of the toxin to the central nervous system the less is the amount required to kill*. The minimal lethal dose is much less if injected into a nerve than if injected anywhere else; it is less when injected intramuscularly than if injected subcutaneously or intravenously.

The *incubation period* depends on the distance from the cord of the site of injection of the toxin. If the toxin is injected into one of the lower extremities of a rabbit, the first definite symptoms appear about the third day; if the injection is made into the subdural space of the cord, or into the cord itself, the incubation period is only a few hours. Division or resection of nerves after intramuscular injection of toxin could save the lives of rabbits only if done within about one hour of the time of injection of the toxin; but even in fatal cases life was prolonged more than when the nerves were not cut, no matter if an interval of from eighteen to forty hours elapsed between the injection of the toxin and section of the nerves of the limb (Sawamura). The use of cocaine in the afferent nerves, or the division of all sensory nerves (leaving the motor nerves intact) may retard the development and diminish the severity of the course of tetanus, but it cannot prevent it. The same is true of intradural division of the posterior nerve roots (Sawamura). If local tetanus has not lasted long, it can be stopped by section of the nerves or by use of curare; but if it has lasted over two days, the tetanic muscle cramp will not be influenced by these methods and will persist even after death. According to Gumprecht this is to be explained as a fatigue phenomenon commonly seen in physiological experiments; he never found reactions of degeneration present.

1. *By what structures of the nerve does the toxin reach the cord?*

Gumprecht (1894) and Stintzing (1898) claimed that it went only through the peri- and endoneurium, passing thence mostly into the cerebrospinal fluid, but also entering directly into the spinal cord, though in smaller amounts. In support

of this theory, Gumprecht found that if he injected toxin into the subdural space of the cord in the lumbar region, local tetanus began first in the animal's hind legs. Stintzing found that the cerebrospinal fluid of two men, very ill with tetanus, was very toxic for mice, while the blood of these patients was not toxic. But Meyer and Ransom (1903) repeated Gumprecht's experiment, and found, if care was taken not to wound the pia or the cord, that general (descending) tetanus was produced before local (ascending) tetanus; moreover, they did not find the cerebrospinal fluid toxic either in patients or experimental animals. These latter investigators (*l. c.*, Obs. 32, S. 413) injected a moderate amount of toxin into the sciatic nerve of a highly immunized rabbit, and nevertheless fatal ascending tetanus developed; yet examination showed that the blood, the cerebrospinal fluid, and the nerve lymph contained active antitoxin. The amount of antitoxin in the nerve lymph, however, was very small—very much less than that in the blood. A twenty-five-fold lethal dose of toxin had been without effect when injected subcutaneously, because at once neutralized by the antitoxin in the circulating blood; two drops of the blood contained more than enough antitoxin to neutralize the fatal dose of toxin injected into the nerve. Their conclusion was that the toxin when injected into the nerve must have reached the cord by way of the axis cylinders, since the antitoxin in the nerve lymph could not neutralize it; but it seems to us an equally correct conclusion that the amount of antitoxin in the nerve lymph was insufficient, even if that in the blood was superabundant. Further reference to this question is made in connection with the prophylactic use of antitoxin (p. 128, footnote).

All that it seems safe to conclude is:

(1) The toxin surely ascends the nerves by way of the axis cylinders (perhaps by means of a centripetal protoplasmic current), and in this way can produce a severe tetanus ascendens.

(2) The transmission of toxin through the peri- and endoneurium cannot be excluded, and the toxin absorbed in this way may play an important part in the development of local tetanus.

The above facts, briefly outlined, are those which prove that the toxin ascends the peripheral nerves to the cord.

2. *The toxin spreads to neighboring parts of the spinal cord.* The diffusion of the toxin in the spinal cord has been demonstrated in several ways. Marie and Morax injected toxin into the lumbar swelling of the cord, and thereafter found toxin in the dorsal cord. If the cord is cut through above the lumbar swelling, tetanus develops only in the lower extremities in cases where toxin has been injected previously into the sciatic nerves, and no tetanus develops in the parts supplied by the cord above the point of section. Moreover, the usual sequence of symptoms in ascending tetanus bears witness to the gradual spread of the toxin within the spinal cord—first the wounded extremity becomes tetanic, then the other lower limb; next the back and abdomen, then the fore limbs, and finally, the neck and head.

Invasion of the sensory portions of the cord also occurs, and is responsible for the extreme hyperexcitability, evidenced by the facility which very feeble peripheral stimuli possess of causing severe clonic convulsions during the disease. If the toxin is injected experimentally into the sensory nerves it is blocked effectually by the ganglia on the posterior roots; if injected into the posterior roots on the proximal side of the ganglia, a very painful form of the disease, termed Tetanus Dolorosus, is produced; and the animals die of exhaustion before the motor phenomena, so characteristic of the disease in its ordinary forms, have had time to develop.

3. *Some of the toxin enters the general circulation.* This is proved primarily by the fact that toxin has been found in the blood on numerous occasions, in experimental animals and in man. It was first found in the blood of man by Nissen

in 1891.¹ *This toxin eventually reaches the cord and produces descending tetanus.* This is proved, because even after all the nerves of a limb have been cut, tetanus descendens can be produced by injecting toxin into the enervated extremity, though many times the usual lethal dose is required. It is certain that the toxin reaches the cord from the general circulation chiefly, if not only, by being brought into contact first with the end-plates of the various motor nerves, and by being transferred through these nerves to the cord. This occurs even when the toxin is injected intravenously, but at present it cannot be certainly proved that no toxin reaches the central nervous system *directly* by the blood; but it seems highly improbable.

MORBID ANATOMY OF TETANUS. Thus far it has not been possible to find, in the peripheral or central nervous system, or elsewhere, any changes which are specific for tetanus. There may be swelling and fragmentation of the axis cylinders, and hemorrhages with vacuolation of the cells in the anterior horns of the spinal cord; but these changes cannot be distinguished from those due to other causes. The recovery of the *Bacillus tetani* from the point of inoculation often is impossible, and scientific confirmation of the diagnosis must rest in many cases upon the reproduction of tetanic symptoms in animals by the injection of toxin extracted from the patient. In the vast majority of cases the clinical diagnosis is quite certain enough without any confirmatory evidence from the laboratory.

Of 15 cases studied bacteriologically, Huber reports that the *Bacillus tetani* was recovered from the wound in 8. This seems a larger proportion of successful cultures than in most series of cases. In our own series of 23 cases, search was made only in a few instances, but never was successful.

In 2 patients, in whom the wound was in the lower extremity, Porter and Richardson excised the inguinal lymph nodes,

¹ According to Stintzing, however, it could not be demonstrated in the blood by Rosenbach, Schulz, Billroth, Moritz, Henoeh, and Engelmann.

and in both cases found tetanus bacilli in the nodes.¹ This demonstrates that the bacillus does not, in all cases, remain in the immediate vicinity of the point of entrance, and suggests that the persistence of symptoms of toxin absorption even after wide excision or amputation of the primary focus may be due to toxin elaborated by bacilli lodged in the anatomically related lymphatics.

CAUSE OF THE SYMPTOMS OF TETANUS. The toxin stimulates the motor cells of the spinal cord, with the result that the muscles controlled by these cells are thrown into tonic spasm; the toxin, as already noted, also renders the sensory side of the cord extremely susceptible to external stimulus, so that very insignificant stimuli, such as the slamming of a door, jarring the patient's bed, a sudden draught of air, etc., will bring on clonic convulsions, or at least will greatly intensify, for the moment, the tonic spasms.

THERAPEUTIC INDICATIONS. These may be summarized as follows:

1. To prevent the development of tetanus.
2. To remove the source which supplies the toxin, *i. e.*, the bacilli of tetanus.
3. To head off and neutralize the toxin already formed.
4. To depress the functions of the spinal cord.
5. To sustain the life of the patient by proper nourishment, nursing, etc.

PROPHYLAXIS OF TETANUS.

It is well known that *certain classes of wounds*, received in certain surroundings, are more often followed by the development of tetanus than are ordinary wounds. The *Bacillus tetani* is anaërobic, and is found especially in garden soil, barnyards, stables, etc. Probably it normally infests the intestinal tract of horses and cattle, and is deposited with their dung. So long as the mucosa of their gastro-intestinal

¹ Schnitzler had found the bacilli in the inguinal lymphatics at autopsy, and Fricker also had found them in the related lymph nodes.

tract is intact, they are not liable to infection by this channel, on account of the antitoxic properites of the intestinal juices, especially the bile (H. Vincent). Matas suggests that post-operative tetanus may be due to tetanus bacilli, latent in the patient's intestinal tract, ingested with uncooked food, and infecting the operative wound by fæcal contact. There is no good evidence that it is due to the use of infected catgut. According to Fox, tetanus bacilli are found in the fæces of 5 per cent. of mankind, and in the fæces of 20 per cent. of men who work about horses.

On these accounts, wounds received by farmers, gardeners, stablemen, etc., are especially liable to be infected with tetanus bacilli. Wounds by farming or gardening implements; wounds by axes, as in felling trees; machinery crushes; wounds produced by dragging in the dust, by kicks or bites of horses; wounds by nails, spikes, etc., incrustated with dirt and rust—these are the causes most to be feared.

In our own series of 23 cases there were 10 in which the wound was contaminated by country earth or street dust; 2 caused by splinters from the floor; 5 by rusty nails or iron spikes; 3 due to machinery accidents; and 1 each to a gunshot wound, puerperal infection, and an explosion.

Inoculation is favored by *anaërobic conditions of the wound*. Especially favorable sites are wounds in which the tissues are sloughing; the best culture medium for tetanus bacilli, according to Tarrozzi, is that which contains some dead organic tissue. Thus punctured wounds, contused and lacerated wounds, and wounds in which there are foreign bodies (earth, machine oil, splinters, wadding, etc.) offer favorable conditions for the development of any bacilli present. A mixed infection, especially with saprophytic bacteria, is favorable because these organisms, being aërobic, absorb all available oxygen and provide anaërobic conditions for the tetanus bacilli.

Care of the wound is naturally the first step in the prophylaxis of tetanus. Such wounds as are considered to offer

favorable soil for the development of tetanus bacilli should be treated with even more than ordinary antiseptic precautions. If the original wound is properly treated, the development of tetanus is unusual. Of our own 23 patients, only 7 had received proper treatment of the wound; and, even in these 7, extra precautions not adopted no doubt would have been taken if the possibility of the development of tetanus had been kept in mind. It is our firm belief that efficient care of the wound as soon as possible after it is received is by all means the most important feature in prophylaxis. We have never yet had a case of tetanus develop in a patient whose wound has been under our care from the first.

Here we must call attention to Case 11 of the present series. This patient was seen by both of us in the Out-patient Department a few hours after his injury was received. Recognizing the gravity of the wound, we referred him at once to the ward of the hospital. Unfortunately he was permitted to return home almost immediately, without proper treatment of the wound, and ten days later developed acute tetanus, from which he died. Efficient treatment of the wound at first might have altogether prevented tetanus, or at all events might have lessened its severity.

Our method of treating a suspected wound is as follows: (1) The surrounding skin is painted with a 3 per cent. alcoholic solution of iodine. (2) Then *all parts of the wound are made accessible*, by wide incision if necessary. (If a punctured wound of the foot, of suspected nature, exists, it is freely opened to its depths, dividing the plantar fascia as far as necessary. If the patient lives far from the hospital, and cannot return home on crutches, he is kept in the ward.) (3) The wound is mechanically cleansed by scissors and forceps, and then is thoroughly swabbed out with the iodine solution. (4) The wound is lightly filled with gauze soaked in the iodine solution, and is properly dressed. We *avoid all caustics*, as they kill the tissues, and the resulting sloughs, even if minute, furnish favorable sites for the growth of

tetanus bacilli. At subsequent dressings (daily at first) the wound is exposed by removal of the iodine gauze, is irrigated with peroxide of hydrogen until active effervescence ceases, and is again filled with gauze soaked in the iodine solution. This method of dressing is continued until healthy granulations are formed. Bockenheimer has concluded from experimental work that the best dressing for these wounds is balsam of Peru, which he believes is antibacterial to the tetanus bacilli. Hessert commends this dressing.

*The Prophylactic Use of Antitoxin.*¹ This was first extensively employed in veterinary practice by Nocard, in 1895,

¹ For the following information we are indebted to Dr. A. Parker Hitchens, of Glenolden, Pa.: The United States Government has established a standard method for testing the strength of tetanus antitoxin: "The immunity unit for measuring the strength of tetanus antitoxin shall be ten times the least quantity of antitetanic serum necessary to save the life of a 350-gram guinea-pig for ninety-six hours against the official test dose of the standard toxin furnished by the Hygienic Laboratory of the Public Health and Marine Hospital Service." The official test dose of toxin is one hundred times the smallest quantity of toxin which will kill a guinea-pig within ninety-six hours. Dr. Hitchens adds that Rosenau and Anderson (1908) found in a German serum which they examined 330 units per c.c., and in some French serums from 40 to 66 units per c.c. Dr. Hitchens examined some French serums in 1910, and found they contained from 30 to 50 units per c.c. He permits us to publish the following results of other examinations made by him in 1910. He thinks that the tests may have been a little too rigid, and that the results noted below are about 15 per cent. too low.

Behring (5 fach.)	220 units per c.c.
Behring (5 fach.)	200 units per c.c.
Berne	12 units per c.c.
Höchst (4 fach.)	110 units per c.c.
Höchst (5 fach.)	250 units per c.c.
Krakau—less than	1 unit per c.c.
Vienna	120 units per c.c.
Lister Institute (veterinary)	30 units per c.c.
Rebman, London, less than	1 unit per c.c.

If the serum at present in use in these countries remains as weak as in these samples, it is evident that the doses of antitoxin administered abroad for therapeutic effect have been quite insufficient. In this country tetanus antitoxin is supplied in tubes each of which holds 10 c.c., the strength being 1500, 3000, or 5000 units per 10 c.c., according to the concentration.

and is found to be almost of absolute value. That it is not by any means always effective when used in man is only too well known. The reasons for the difference in its action in horses and in man must depend, as pointed out by Solieri, on one of two factors: either it is not so useful or it has not been properly administered. (1) Perhaps the fact that horses (in which its use is most successful) are treated by antitoxin derived from their own serum, while human patients are treated by an alien serum (horse), makes the difference. But to this supposition (and it is little more) it may be replied that other animals, such as cattle and sheep, are well protected by the horse serum. (2) The most reasonable explanation of the numerous failures of antitoxin to prevent the development of tetanus in man lies in the faulty manner in which it has been administered, in most cases. Remertz has tabulated 55 cases where tetanus developed in spite of the prophylactic use of antitoxin; in 38 of these cases the diagnosis is positive; and as only 30 of these patients died (53 per cent.) it is evident that the antitoxin at least rendered the disease less fatal; but even with all allowances the mortality in these cases cannot be brought below 40 per cent.

There are three things to be considered in the matter of the prophylactic use of antitoxin: (1) The frequency with which it should be given. (2) The site of the injection. (3) The quantity to be administered.

1. *The frequency of prophylactic injections.* It appears to be a well-ascertained fact that the antitoxin is all eliminated from the system in about eight or ten days after the injection. It remains in practically undiminished amount for a week, and then rapidly diminishes to nothing in the course of two days (Dehne and Hamburger). As the tetanus bacilli, if any are lodged in the wound, may not begin to produce any toxin until at least as long a time as this has elapsed, it is manifestly important, if the antitoxin is to be of use, that some should be present in the system for two or three weeks after the receipt of the injury. In many cases the

tetanus bacilli do not enter the wound at the time of the accident, but are introduced secondarily by the carelessness of the patient in neglecting to return for dressings, and in attempting to care for the wound in septic surroundings at home; in such cases surgery can scarcely be held responsible. But even if present in the wound from the first, the bacilli may lie dormant for weeks in spore form, before development occurs with the production of toxin.

Vaillard cites 6 cases in which the period of incubation was over a month, in 1 of which (Terrier) it was eighty-seven days. In Case 4 of our series, with an incubation period presumably of eight weeks, the onset was very gradual, but the disease then developed in frightfully acute form and was rapidly fatal. Fox says that tetanus spores injected into a guinea-pig have remained latent for four months, and have then, without apparent cause, produced an outbreak of the disease. In some such manner as this, no doubt, are to be explained the not very unusual cases where the symptoms of tetanus recur some time after apparently complete recovery from a frank attack of the disease. Fink reports two severe relapses at intervals of several months, due to the development of latent spores; Fedden records such a case, where symptoms recurred over one month after complete recovery; and Reynier narrates a rather mysterious case where slight recurrent attacks of tetanus occurred throughout a period of six years, and in which finally a typical and very severe attack occurred, apparently caused by the hypodermic injection of a quinine salt.¹

The necessity for a second injection of antitoxin about the eighth or tenth day is thus very evident, and it may be well

¹ Subcutaneous or intramuscular injections of quinine salts may arouse latent tetanus spores, because they cause local necrosis (Vincent, 1904). This subject is well discussed editorially in the *Indian Medical Gazette* (1911, xlvii, 141 to 142). This teaching is controverted by Palmer and by McCampbell; the latter holds that quinine can be held responsible only if there is already present a secondary (pyogenic) infection.

to administer a third injection during the third week. Solieri insists that this injection must be renewed every eight or ten days until all necrotic tissues are removed and there is a clean granulating surface. In almost all of the 55 cases, noted above, in which tetanus developed in spite of the prophylactic use of antitoxin, *the injection was not repeated*.

But that even repeated injections may prove ineffectual is demonstrated by the well-known case recorded by Maunoury: 10 c.c. of antitoxin was injected at once after the injury; this was repeated in six days; and again six days later a third injection was given. Twelve days after the third injection, as the patient complained of severe pain in the wounded part, a fourth injection was given, and the limb was amputated. In spite of this, three days later (twenty-seven days after injury) tetanus developed and the patient died of severe tetanus in twenty-four hours. (Was this possibly a case in which some bacilli had lodged in the lymph nodes, or was it that the antitoxin injected at the end of the second period of six days had all escaped from the body at the end of the next twelve days, when the fourth injection was given?)

2. *The site of the injection.* In most of the reported cases no details of the site of injection are given, but in such cases it is presumed that the antitoxin was administered subcutaneously.

Calmette, and later McFarland, applied antitoxin in powdered form directly to the wound, in experimental prophylaxis, and found it of great value. Lop, however, found it did not prevent the development, eight days after injury, of tetanus fatal in three days, though the wound was powdered with antitoxin one-half hour after the injury, and again three days later. Luckett had a similar experience. It may be, as suggested by Tuffier, in the discussion of Lop's case, that the antitoxin was absorbed by the dressings applied instead of by the wound. Remertz refers to two other cases in which the use of powdered antitoxin proved ineffectual.

The subcutaneous use of antitoxin in prophylaxis is open

to the same objections raised against this method of therapeutic administration (p. 133). In any circumstances the antitoxin should be given as near to the wound as possible, so as to flood the tissues in the immediate vicinity; and the injection should be deep, intramuscular if possible, so as to permit its rapid absorption by the motor nerves. If the antitoxin can reach the peripheral nerves before any toxin that is formed reaches them, the likelihood that the toxin ever will reach the spinal cord is much decreased. If any nerves are exposed in the wound the antitoxin should be injected into them.

This was the idea of Meyer and Ransom, who thought the imperilled spinal-cord centres might be protected from the toxin by blocking the afferent nerves with antitoxin. They proved that if one sciatic nerve of a rabbit was injected with antitoxin, and simultaneously both legs below the knees were injected with toxin, then only the protected limb remained free from tetanus or developed it very late. Sawamura repeated these experiments: first he injected twice the lethal dose of toxin in the leg, and from sixty to ninety minutes later injected antitoxin into the sciatic nerve of the same limb; 6 out of 8 rabbits thus treated did not die, and the limb so treated remained free from local tetanus in all, even in the 2 fatal cases where death was caused by general tetanus. The 6 control animals (not protected by antitoxin in the nerve) all developed local tetanus, and 2 died of general tetanus.¹ If antitoxin was injected subcutaneously or intravenously, even in two or three times the quantity, it was powerless to prevent the development of local tetanus. The experiments conducted by v. Graff, to test the prophylactic

¹ The fact that intraneural injections of antitoxin block the further progress of toxin along the nerves, is good evidence, it seems to us, either that the toxin ascends in large amounts through the lymph channels of the nerves, or that antitoxin can be readily absorbed by axis cylinders which are in its neighborhood. It evidently is not necessary for the injecting needle to penetrate each and every axis cylinder of the nerve trunk.

value of antitoxin administered intravenously, are detailed at p. 135.

3. *The quantity of the injection.* Usually 1500 units is the amount employed as a prophylactic dose. Remertz thinks that even if the injection is made very soon after the injury, the least amount that can be useful as a prophylactic dose is from 2 c.c. to 5 c.c. (=20 A. E.); according to Hitchens' table (p. 124) this is equivalent to about 1500 units, U. S. A. Remertz says that Lotheisen thought 100 A. E. was the proper prophylactic dose. Taking the average weight of persons injected as 150 pounds, and figuring out the potency of the antitoxin from the known value of a unit (U. S. A.), we find that 1500 units is a little over two thousand times the amount necessary to neutralize the minimal lethal dose of toxin in such an individual. Such an amount is by no means excessive, in view of the fact that the amount of antitoxin required to prevent death increases in geometrical progression with the lapse of time. After the lapse of one hour, twenty-four times as much antitoxin is required as when antitoxin and toxin are injected simultaneously (Remertz). Certainly antitoxin will be more useful if injected in the immediate neighborhood of the wound than elsewhere, as it will then be in more concentrated form as it comes into relation with the forming toxins.

Objections to the prophylactic use of antitoxin. The chief objection is the expense. Unpleasant symptoms from its use are scarcely known, even when it has been used in massive doses as a therapeutic measure. The site of the injection often is painful, and if carelessly given, harm may result.

We are cognizant of one case where the injection was made by a trained nurse, by order of a physician. The needle was introduced on the outer side of the thigh, but too near the knee; the result was that the antitoxin was injected into the subfemoral bursa, and the patient was laid up in bed for several days, with "water on the knee."

Occasionally, in susceptible individuals, an erythematous

or urticarial rash develops and multiple arthritis has been reported (Reynier), but other evidences of anaphylaxis do not appear to have been observed.¹

In conclusion, it may be said that the worst reproach that can be made against the prophylactic use of antitoxin is that, while it is harmless when carefully administered, it may be useless. That it never is useful has not been proved; and we believe that so long as a possibility of its usefulness exists, it is proper to employ it in the case of suspected wounds. In none of the patients in our series had a prophylactic injection been given. At least 1500 units should be given in the immediate neighborhood of the wound; it should be injected deeply into the muscles; or if possible, into the nerves (when a smaller quantity will suffice). This injection *should be repeated* at the end of seven or eight days; and, if possible, a third injection should be given during the third week.

Finally, a few words may be said about "*Fourth of July Tetanus*." In spite of diligent, and we believe unprejudiced, study of the subject, we have been unable to convince ourselves that such injuries received on "the day we celebrate" are more liable to be followed by tetanus than other contused or lacerated wounds.

During the years one of us was on duty at the Children's Hospital, Philadelphia, where many such cases were treated every year, antitoxin never was employed as a prophylactic (the hospital being unable to afford it), but the wounds were cleansed as above described with most meticulous exactitude; yet not one case of tetanus due to such an injury ever occurred. Indeed, we have never seen anywhere a case of tetanus due to such an injury.

We believe, however, that the campaign for the use of antitoxin as a prophylactic has accomplished a marked

¹ Riche has recorded a death following the prophylactic injection of tetanus antitoxin in a patient under the care of Rilhac; but the *cause* of death was undetermined.

reduction in the incidence of tetanus; but we hold that this result may be attributed as rationally to better care of the wound as to the antitoxin employed, because a physician who thinks of antitoxin thinks also of tetanus, and the thought of possible tetanus impels him to take proper care of the wound. Moreover, in only an extremely small proportion of cases has the injection been repeated.

TREATMENT OF TETANUS

A. The first logical step in the treatment of this disease is the *removal of the source which supplies the toxin*—that is, of the tetanus bacilli. If the point of inoculation is known, it should be attacked directly. The wound should be opened widely, and should be mechanically cleansed of foreign bodies and sloughs. Then it should be swabbed out with a 3 per cent. alcoholic solution of iodine, rinsed with hydrogen peroxide, and filled loosely with gauze soaked in the iodine solution. We believe all caustics should be avoided, as favoring the growth of tetanus bacilli by the formation of sloughs. If the nature of the case demands it for other reasons, amputation should be done; then the stump should be left open, and treated as the original wound. Probably in many cases it will be well to follow Porter and Richardson's suggestion to excise the related lymph nodes, particularly if they are palpably enlarged.

B. The next indication is to *head off and neutralize the toxin already formed*. This involves a discussion of the THERAPEUTIC USE OF ANTITOXIN (see footnote, p. 124).

Before the introduction of antitoxin as a therapeutic measure the mortality was from 80 to 90 per cent. (Rose, 1897). Collective statistics of cases treated with antitoxin (1897 to 1903), according to Sawamura, showed a death rate of from 28 to 45 per cent. Of course, a collection of isolated case reports is apt to give too low a mortality, owing to the fact that surgeons who have had successful cases will report

them, while the larger number do not report their unsuccessful cases. But the fact that no better results were reported from the therapeutic use of antitoxin in tetanus, in view of the remarkable successes from the use of diphtheria antitoxin, was explained on the theory that the antitoxin had not been used early enough. In 1902, Ullrich, according to Sawamura, remedied this apparent defect by collecting 13 cases in which antitoxin had been used early; but only one of these patients recovered; and of 54 collected cases, in which it was used early (subcutaneously), between 1899 and 1905, Busch (Sawamura) found the mortality was 66.5 per cent. In the elaborate statistics collected by Jacobson and Pease the mortality in 191 cases in which antitoxin was used, was 69.6 per cent.; these statistics are especially valuable because they represent series of consecutive cases from various sources. Fricker has published statistics of 40 consecutive cases; in the first 18 (1889 to 1897), no antitoxin was used, and 16 deaths occurred (88.8 per cent.); in the last 22 cases (1897 to 1902), antitoxin was used, and the mortality was only 55.5 per cent. In spite of this decrease in mortality, many physicians have denied that antitoxin has any therapeutic value in acute severe cases.

Huber has reported recently in great detail 69 cases of tetanus treated in Sauerbruch's clinic at Zurich, between 1881 and 1911. Of this whole number, 18 patients recovered and 51 died, a mortality of 74 per cent. Before 1900 antitoxin was not used, and of 31 patients treated without it, 20 died, a mortality of 64.5 per cent. Antitoxin was used in all of the 38 patients treated since 1900, but of these only 7 recovered, while 31 died, a mortality of 81.5 per cent. Huber thinks all of these 7 patients would have recovered even if no antitoxin had been given. But the total amounts used were small (the greatest amount used in any one case was 150 c.c. = 375—750 A. E.), and the mode of administration is open to criticism; this will be discussed later (p. 142).

But here, again, as in the question of its prophylactic use,

we must inquire as to the frequency, the quantity, and the site of the injections. In most of the isolated case reports the antitoxin has been injected only once or twice, in small quantities, and subcutaneously.

Exactly how the antitoxin acts upon the toxin is not known. According to Solieri, the idea that its neutralizing action is a direct chemical one, as that of an acid on an alkali, has been abandoned. Dönitz taught that it could not only neutralize the toxin circulating in the blood, but also could loosen toxin already bound in the central nervous system and drive it out of its almost impregnable fortress. According to Metschnikoff, tetanus antitoxin stimulates phagocytosis. From numerous experiments Kraus and Amiradzibi conclude that antitoxin cures by drawing the toxin out of the cells where it is lodged, but does not itself enter into the toxin-containing cells. Recovery under the use of antitoxin they think depends on the possibility of this diffusion; and as such diffusion can take place experimentally through a membrane of collodion and through reeds (Schilfröhren), they think it is not necessary in the human body for antitoxin to be in direct contact with toxin for it to exert its neutralizing influence on the latter.

I. SITE OF INJECTION OF ANTITOXIN. The following sites of injection have been advocated: Subcutaneous, intravenous, intraspinal, intraneural, intracerebral, and intramuscular.

1. *Subcutaneous Injections.* This is the method usually employed, but, as already noted, the reports are not encouraging. If administered in this way, the antitoxin is absorbed by the lymphatics, transported to the veins, passes through the lungs, and finally is distributed through the arterial system to all parts of the body. Only a homeopathic dose ultimately reaches the motor nerves through which the toxin is being carried to the spinal cord, while by far the greater part of the antitoxin is distributed to the viscera, where it can be of no possible use. Administered in this way, overwhelming amounts are required to produce any effect, and it is evidently the height of extravagance so to employ it.

The manufacturers of antitoxin recommend the administration of from 15,000 to 18,000 units (subcutaneously) every three hours; and such an amount (120,000 to 144,000 units) in the course of twenty-four hours is not unreasonable when it is recollected that only a very small fraction of what is administered subcutaneously can be expected to reach the seat of disease, while the rest is a shocking waste of a valuable and very costly remedy. For the amount mentioned (144,000 units) the cost is about \$100, even with the discount allowed to hospitals. (One dozen tubes of 1500 units each (18,000 units) cost the Episcopal Hospital, \$12.67; eight doses of 18,000 units cost \$101.56). Note the history of Case 23, of our series; here antitoxin was administered subcutaneously in appropriate doses (99,000 units on the first day; 65,000 units on the second day; 60,000 units on the third day)—to a total amount of 224,000 units,¹ and recovery followed. The cost to the Hospital, with discounts deducted, was about \$180. The patient was a very poor man, a charity case; but when told of the expense of his treatment, he and his friends collected \$10 and donated that sum to the hospital, as a "Widow's Mite."

Compare with this history the case of a severer case of tetanus (Case 11 of our series): On the first day 3000 units were administered intraspinaly; on the second day, 750 units were injected into the sciatic nerve, and 750 units deeply into the tissues around the wound. The patient recovered, and the total cost to the hospital was about \$3.

The quantities of antitoxin used in each case were logically correct, but only in Case 11 was the mode of administration rational.

¹ The nearest approach to this total quantity that we can find reported was administered to a patient under the care of Beates and Thomas. The most given on any one day was 97,940 units, the total amount being 213,700 units; but as the patient was a boy of fourteen years (weight 130 pounds), this amount may be relatively greater. Neary administered 280,000 units to his patient, who recovered; but as this quantity was spread out over a period of two weeks (10,000 units every twelve hours), it is not comparable to the doses above mentioned.

2. *Intravenous Injections.* The effects of intravenous injections of antitoxin have been studied experimentally by v. Graff, both as regards prophylactic and therapeutic use:

(a) In a first series of experiments he administered the toxin by *intramuscular* injection. (a) Antitoxin was administered to 14 rabbits *before* toxin was injected; of these animals only 4 died, and not one had positive signs of tetanus. (b) Antitoxin (intravenously) and toxin (intramuscularly) were administered *simultaneously* to 8 rabbits; 4 of these died, but only 1 presented symptoms of tetanus. (c) Toxin was administered intramuscularly to 8 rabbits, and *from fifteen to eighteen hours later* antitoxin was injected intravenously; 4 of these rabbits died, 2 with only local tetanus, and 2 with general tetanus.

(b) In a second series of experiments he administered the toxin by *intraneural* injection. (a) Toxin was injected intraneurally in 6 rabbits; all died of tetanus in from thirty-six hours to five days. (b) Antitoxin was administered intravenously to 19 rabbits *from six to ten hours before* a lethal dose of toxin was injected intraneurally. Only 2 of the animals lived and 17 died after intervals of from five to twenty-five days; but in the fatal cases death was attributed to intercurrent gastro-enteritis, no evidence of tetanus having been present. (c) Antitoxin was injected intravenously *twenty-four hours before* toxin was injected intraneurally; of 5 rabbits so treated none developed tetanus, 2 lived, and 3 died from no apparent cause in from four to nine days. (d) Antitoxin (intravenously) and toxin (intraneurally) were injected *simultaneously*; of 5 rabbits so treated none developed symptoms of tetanus, yet all but 1 died in less than eight days. (e) Toxin was injected intraneurally, and *from fourteen to eighteen hours later* antitoxin was injected intravenously; of 3 rabbits so treated, the first developed local tetanus, but recovered, only to die on the ninth day of purulent peritonitis; the second developed local tetanus but recovered; the third developed local tetanus and then general tetanus, and died on the

fifth day of general tetanus in spite of intravenous injections of antitoxin repeated every day. (f) Toxin was injected intraneurally, and *from fifteen to seventeen hours later* antitoxin was administered intravenously; of 7 rabbits so treated, 4 died as if no antitoxin had been given; in 1 life was prolonged by the treatment until the sixth day, when death from general tetanus occurred; and in 2 recovery ensued, although very severe general tetanus had already developed. These 2 recoveries, v. Graff thinks, are to be explained only on the ground that the antitoxin was able to neutralize toxin already absorbed by the nervous system.

The conclusion of v. Graff is that intravenous administration is the most effective method, but that subdural (intraspinal) administration holds next rank, because he believes in this way antitoxin is rapidly discharged from the cerebrospinal fluid into the blood.¹ Intraneural injections of antitoxin were not employed by v. Graff, but the results of Sawamura's experiments, detailed below (p. 140), as to the relative therapeutic value of intravenous and intraneural administration of antitoxin, seem to demonstrate conclusively the superiority of the intraneural method. From these experiments, to be discussed presently, it is evident that antitoxin administered only by the intravenous method is not very effective; and the same objections as to extravagance apply to this as to the subcutaneous method, only in less degree, because the antitoxin reaches the circulating toxin in shorter time, and can exert its influence sooner on the toxin already absorbed by the peripheral nerves or the spinal cord, if only it is used in sufficient quantities.

That the amount required to produce any therapeutic effect is immense, is proved by the observations of v. Leyden; he neutralized all the toxin in the patient's circulating blood rendering it harmless to experimental animals; but his patient died of tetanus. And in a patient under the care of Blumen-

¹ v. Graff says that L. Simon has treated 6 consecutive cases of tetanus by intravenous injections of antitoxin, with 4 recoveries.

thal and v. Leyden, they succeeded in neutralizing almost entirely the circulating toxin, but much toxin remained in the cerebrospinal fluid. More recent observations tend to show that the amount of toxin circulating in the blood rapidly diminishes after the first few days of the disease, while that in the nervous tissues steadily increases.

The following advantages of intravenous administration are pointed out by v. Graff:

- (i) The antitoxin gets into the blood as soon as possible.
- (ii) This method can be used even when the point of inoculation is doubtful, or unknown, or inaccessible. He recommends it especially for cases of puerperal origin.
- (iii) It is easier than intraspinal or intraneural injection, is less painful, and can be repeated.

As regards the first advantage, it may be replied that it is more important to have the antitoxin in the nerves and spinal cord than in the blood. Intravenous administration is not the only available method for cases mentioned in the second paragraph, since intraspinal (subdural) injections may be made; and these, as well as intraneural injections, can be repeated as often as is desirable. However, they are more painful to the patient, sometimes requiring a general anæsthetic, and intraneural injections at any rate require accurate anatomical knowledge and surgical skill.

3. *Intraspinal (Subdural) Injections.* These were originally suggested by Jacob; but he came to the conclusion, after some experimental work, that they were useless, because he thought all the antitoxin so administered rapidly escaped into the general circulation. Sicard, however (quoted by Hofmann), obtained better results in his experiments with dogs. The method was first used successfully in 1899 by v. Leyden, who thought this the best method of all, emphasizing the fact that the antitoxin thus came into intimate relation with blood- and lymph-vessels of the cord, and so was conveyed more quickly to the medullary cells. Whether or not antitoxin injected into the subdural space of the cord

acts directly upon the cord itself, or upon the nerve roots, does not appear to have been determined. Certainly it has not been disproved, and if reasoning by analogy with tetanus toxin be allowed, it is altogether probable that antitoxin injected intraspinally is in large part absorbed into the nerve roots and the cord itself, especially if the pia or the nerve tissue is punctured. Mere withdrawal of cerebrospinal fluid by lumbar puncture has been suggested as a therapeutic measure, and it is possible that the rapid decrease in severity of the symptoms often seen may be produced in this way. It is not reasonable to expect the specific action of the antitoxin to be manifested for several hours.

Though, experimentally, treatment of tetanus by intraspinal injections of antitoxin has not been very encouraging, much more satisfactory results have been obtained in actual practice.

Hofmann reported from v. Hacker's clinic, at Graz, a series of 30 consecutive cases of tetanus. From theoretical considerations they were opposed to the intraspinal administration of antitoxin, and in the first 13 cases only subcutaneous injections were used; of these patients 7 died, a mortality of 53.8 per cent. In 3 of these cases, 2 of them fatal, carbolic acid was also employed subcutaneously. In a fourteenth patient, who also died, both subcutaneous and intraneural injections were given. Of the cases treated by subcutaneous injections only, 5 came under treatment within thirty hours of the onset of symptoms; and of these patients (mostly acute cases), 4 died, a mortality of 80 per cent.; 8 cases came under treatment more than thirty hours after the onset of symptoms, and of these only 3 patients died, a mortality of 37.5 per cent. When the fifteenth patient came under treatment, intraspinal injections of antitoxin were tried, because other treatment did not control the disease, which was of the acute type, and very severe. This patient recovered. Then in the succeeding 15 patients antitoxin was always given intraspinally; and *of the entire series*

of 16 patients treated by antitoxin subdurally, only 2 died, a mortality of 12.5 per cent.; 4 of these were very acute cases, yet all recovered. In 2 cases of this latter series antitoxin was injected intraneurally as well as intraspinally.

Rogers, in a series of 7 acute cases, employed intraspinal as well as intraneural injections, and in administering the intraspinal injections he endeavored to inject at least some of the antitoxin directly into the spinal cord (intramedullary injection), so that it might reach the nerve centres as quickly as possible. Of his 7 patients, only 3 died. Of 5 patients under the care of Lockett, all the 4 who were treated by intraspinal injections recovered, while the fifth patient, not so treated, died.

In our own series of cases intraspinal injections of antitoxin were employed only in 7 patients, of whom 4 died, a mortality of 57 per cent.; but in 1 fatal case the injection was made only three hours before death, as a last resort (Case 2); in another, death was caused by a subsequent overdose of magnesium sulphate, injected into the subdural space (Case 19); and the 2 other deaths occurred in extremely acute cases (Cases 12, 13). In all the successful cases improvement was rapid (Cases 8, 10, 11).

4. *Intraneural Injections.* This method was first employed clinically by Küster in 1902. Jacobson and Pease suggested that the pressure, rather than the specific action of the antitoxin, blocked further absorption of the toxins; but an experiment in which Sawamura employed salt solution instead of antitoxin for intraneural injection, seems to disprove this supposition.

As it is a well ascertained fact that most, possibly all, of the toxin reaches the spinal cord only by travelling up its nerves, it is theoretically logical to inject the antitoxin into the nerves, in order that, like the toxin, it may reach the spinal cord and rout the enemy by the easiest road. That it will do this, when injected intraneurally, admits of no doubt in view of the overwhelming experimental evidence on the

point. Just how the antitoxin acts, and by which intraneural route (axis cylinders or endo- and perineurium) it reaches the cord, has not been determined. Now, though most of the toxin ascends the nerves leading from the wounded part, other lesser amounts of the toxin are simultaneously invading the cord through all the motor nerves of the body; and it is manifestly impracticable to expose and inject antitoxin into all of these nerves. Moreover, when the site of inoculation is doubtful, or unknown, uncertainty must exist as to which nerves should have injections of antitoxin. The only methods we possess for reaching all the nerves at once are (1) intravenous injections, and (2) intraspinal injections. In no case, therefore, should the surgeon depend upon intraneural injections alone, and in no case should he omit either intraspinal injections or injections into the motor nerves leading from the site of inoculation. Especially if the point of inoculation is in a muscular part, or in the upper extremity, will the main bulk of the toxin reach the cord by the motor nerves of the inoculated part; and in such cases particularly are intraneural injections requisite.

Sawamura conducted experiments to determine the relative therapeutic value of antitoxin administered *intravenously* and *intraneurally*. (1) In a first series of 4 rabbits he administered antitoxin *intravenously from eighteen to nineteen hours after* toxin had been injected intramuscularly; all 4 rabbits died of tetanus. (2) In a second series of 6 rabbits he administered antitoxin either *intraneurally alone or intraneurally as well as intravenously, from eighteen to twenty-four hours after* toxin had been injected intramuscularly; and although the *total amount of antitoxin* was no greater or even less than that which had been employed in his first series, yet only 2 of the rabbits died, and these deaths did not occur until the twelfth and the sixteenth days, in cases where the total amount of antitoxin was small, where it had been injected late, and where it had been given only intraneurally, and not intravenously as well. (3) In a third series of 3 rabbits he administered

antitoxin *intravenously* forty hours after the intramuscular injection of toxin, and seventeen hours after the appearance of the first symptoms of tetanus; he administered antitoxin in large amounts *intravenously*, *intramuscularly*, or *subcutaneously*, or *by all three methods*, and *on several subsequent days*; yet *all these rabbits died of tetanus in from nine to fourteen days*. (4) In a fourth series of 4 rabbits, he administered a *much less amount of antitoxin intraneurally*, after the same interval since the intramuscular injection of the toxin as in the third series; yet *not one of these four rabbits died*. In another rabbit, similarly prepared, for the sake of a control experiment, he injected salt solution into the nerve instead of antitoxin; but this rabbit died of tetanus on the ninth day.

Sawamura says (p. 85): "To increase the value of intraneural injections of antitoxin, one must strive not only to neutralize the toxin in the peripheral nerves or that which is later to reach them, but also to bring the action of the antitoxin to bear on the toxin already in the spinal cord; and, therefore, the injection should be made into the nerve as near the cord as possible; and centrally from this point as large an amount of antitoxin as is possible must be injected." He thinks that if in his earlier experiments (the second series quoted above) he had injected the antitoxin into the central instead of into the peripheral part of the nerves, he would have had still better results.

There are few clinical reports of cases treated by intraneural injections of antitoxin; most of them are isolated cases, and hence are of little value. Küster's patient, the first so treated, recovered. Rogers has employed this plan in 7 cases, with 4 recoveries and 3 deaths, a mortality of 43 per cent. (In all these cases intraspinal injections were employed also.) Sawamura, in 1909, collected 12 isolated cases of tetanus, treated by intraneural injections of antitoxin, with only 4 deaths, a mortality of 33 per cent. In our own series of cases it was employed only twice (Cases 11 and 13), only the first patient recovering.

In the patients who have recovered after this treatment, no disability appears to have been caused except in Küster's case, where some neuritis and muscular atrophy and contractures occurred; and these may have been due to the effects of the original injury and not attributable to the treatment employed. Our own patient suffered no inconvenience from the injection made into his sciatic nerve.

5. *Intracerebral Injections.* These were originated by Roux and Borrel, who found the method valuable in experimental work: of 45 guinea-pigs thus treated, 35 recovered; whereas of 17 treated by subcutaneous injections of antitoxin only 2 recovered. But when applied to mankind, the results have been absolutely bad—not only has the mortality not been reduced, but lasting cerebral lesions have been produced in some of the patients who recovered. In 88 cases collected by v. Graff, in which intracerebral injections were used, there were 71 deaths, a mortality of over 80 per cent. Sawamura quotes the following statistics for intracerebral injections: Lambert, 52 cases, 63.43 per cent. mortality; Lereboullet, 26 cases, 67.5 per cent. mortality; Steuer, 55 cases, 67.3 per cent. mortality.

6. *Intramuscular injections* have not been employed in any large series of cases. Antitoxin injected around the wound, whether as a prophylactic or as a therapeutic agent, should be given into the muscular tissues whenever possible, to afford it a readier access to the motor nerves. As a therapeutic method intramuscular injection is better than the subcutaneous, but is perhaps inferior to the intravenous, and certainly is inferior to the intraspinal and intraneural methods.

II. FREQUENCY OF INJECTION OF ANTITOXIN. The series of cases reported by Huber has already been referred to. He thinks the antitoxin employed was of no value, and when we come to study his case reports it is quite evident that in most cases this is true. But it is true because antitoxin was not given often enough to be of any service, as well as because

the total quantities were absurdly small, in view of the fact that most of it was injected subcutaneously. In 17 cases one injection alone was given, and 1 patient recovered. In 16 cases two injections were given, and 4 patients recovered. In 2 cases three injections were given, and none recovered. In 3 cases four injections were given, and 2 patients recovered. Nor is it true that multiple injections were not given because the patients did not survive long enough. Even a patient who dies in twenty-four hours after coming under treatment lives long enough to receive at least eight subcutaneous injections. When it is administered intravenously larger amounts can be given at one time, and one or at the most two injections in twenty-four hours should suffice. Intraspinal injections seldom are requisite more than once daily, and often only every third day. Intraneural injections can be repeated daily if required; this was done on three consecutive days in Case 13 of our series, but unfortunately without a successful result.

III. QUANTITY OF ANTITOXIN INJECTED. No matter what the method of injection, *the most important thing is to get the maximum quantity of antitoxin indicated into the patient's body as soon as possible*. Delay even of a few hours may determine a fatal result; 25,000 units given within the first three hours almost certainly are of more use than 50,000 units given after six hours, or given in divided doses. If one determines to use antitoxin at all, he should, we believe, make it a rule to administer it as early as possible, and *to administer the total quantity indicated as nearly as may be all at one time*. There can be scarcely any doubt that in most of the reported cases, as for example in Huber's series, the amount of antitoxin administered has been utterly inadequate.

As already pointed out, if the injections are given *subcutaneously*, immense quantities are indicated. For an adult, with the usual acute type of case, at least 100,000 units are required in the first twenty-four hours; though a less amount may be sufficient for a child or for a comparatively mild

case, one cannot be sure of the fact, and it is better to give too much than not enough. Administered *intravenously*, a less amount is sufficient; how much it is difficult to say. Probably 15,000 to 25,000 units should be administered at first, and if no effect is apparent, or if the good effect wears off, a similar amount should be given after the lapse of eighteen to twenty-four hours. If given *intraspinally*, from 3000 to 10,000 units should be given, according to the severity of the case; this need not, as a rule, be repeated in less than eighteen to twenty-four hours. Even when administered *intraspinally* a certain interval must elapse before the effect of the antitoxin can be apparent. *Intraneural* injections should be made in as great amounts as the nerves will absorb. We have injected 1500 units into the sciatic nerve, all at once, on several occasions, and 750 units into each of the anterior crural and obturator nerves. If the injections are slowly made practically all of this quantity can be introduced among the nerve fibers.

CARBOLIC ACID INJECTIONS IN THE TREATMENT OF TETANUS. This method was first brought to the attention of the profession in 1893 by Bacelli, who had employed it since 1888. The well-known anæsthetic properties of phenol indicate that it has an affinity for nervous tissue, and it had been used successfully by Bacelli and others in cases of neuritis before he adopted it in cases of tetanus. According to Imperiali, carbolic acid is both antibacterial and antitoxic to *Bacillus tetani*, and acts, moreover, as a nervous sedative. He has collected 190 cases treated by Bacelli's method, with 157 recoveries and 33 deaths, a mortality of only 17.3 per cent. He classified the cases thus:

		Mortality
Severe cases,	94, with 2 deaths and 92 recoveries. .	2.1 per cent.
Severest cases,	39, with 17 deaths and 22 recoveries. .	43.5 per cent.
Fulminating cases,	15, with 14 deaths and 1 recovery . .	93.3 per cent.

These statistics are open to the usual objections applicable to collected cases; and it is strange that among so large a

number of case reports no cases appear which may be classed as mild or chronic in type. It may be that the Italians, recognizing that everything is comparative, class as severe those cases which we consider mild, on the theory that even a mild attack of tetanus is a severe disease. Imperiali, however, quotes Meoni as having observed 5 cases of tetanus in the past four years, all treated by carbolic injections, and with only one death. Surgeons in other countries, however, have not reported as successful results. Pearce Kintzing, almost alone in this country, reports favorable results; he treated 7 patients by carbolic injections, and all recovered; in 3 of these cases the onset of the disease was acute, in 3 the onset is not described, and in 1 the onset was very gradual. In less than half of his cases could the disease be considered very severe in type.

Bacelli's plan is to administer 1 c.c. of a 1 per cent. solution every few hours, preferably into the muscles along the spine, until 80 or 100 centigrams are given in twenty-four hours.¹ In none of the cases mentioned by Imperiali did the total amount administered in twenty-four hours exceed 50 centigrams. Kintzing made a 10 per cent. solution by dissolving the deliquesced crystals of phenol in sterile water; the full adult dose was 10 drops of this 10 per cent. solution (equivalent to 1 grain of pure phenol), which was diluted in 30 or 40 minims of sterile water, just before being injected. Most physicians have preferred to use a weaker solution (0.5 per cent.), and to make the injection every one or two hours, carefully watching for constitutional symptoms of carbolic acid poisoning. The tolerance of tetanus patients for carbolic acid is amazing.

Phenol injections were employed in only one patient in our series (Case 20), in conjunction with small amounts of

¹ Most preparations of antitoxin in this country have added, as preservative, 0.5 per cent. of tri-kresol (Hitchens). Some of the foreign sera are said to contain 0.5 per cent. of phenol. Any therapeutic effect this may have must be beneficial in cases of tetanus.

antitoxin subcutaneously. It was a mild, chronic type of case, of puerperal origin, and though the patient survived the disappearance of all tetanic symptoms for a period of nine days, she eventually died from puerperal sepsis.

Camus has made a series of experiments to determine the comparative value of carbolic acid, magnesium sulphate, and antitoxin in the treatment of tetanus. Dogs were used, and the animals in each series received exactly the same amount of the same toxin, at the same time. These investigations show that magnesium sulphate (intraspinally) and carbolic acid (subcutaneously) have no influence on the evolution of tetanus, no matter in what amounts or at what stage of the disease they are administered. Magnesium sulphate has no other action than as a spinal depressant; and while carbolic acid possibly may have some antibacterial action, it has no effect on the fixed toxin nor on the toxin in course of fixation. Antitoxin, alone, injected simultaneously into the cerebrospinal fluid by lumbar puncture, intravenously, and subcutaneously, *gave very much better results*.

CHOLESTERIN INJECTIONS IN THE TREATMENT OF TETANUS. Experiments having shown that it is the cholesterin of the central nervous system for which tetanus toxin has special affinity, it occurred to Almagia and Mendes that hypodermic injections of this substance might neutralize the toxin. In 2 patients they used successfully injections of from 15 to 30 centigrams of cholesterin in 10 c.c. of distilled water; Pribram, however, reports the use of cholesterin in 3 patients, all of whom died. Among other substances which have been found experimentally to be capable of neutralizing tetanus toxin is urea (Sewaki).

C. The third indication in the treatment of tetanus is *to depress the functions of the spinal cord*. So far we have considered only methods to check the supply of toxin and to neutralize the toxin already formed. Now we come to an equally important factor, because even if the supply of toxin can be stopped promptly, and even if the toxin not yet

firmly bound to the nerve tissues can be completely neutralized, there is in almost every case a large amount of toxin which has become impregnably entrenched in the central nervous system, particularly in the spinal cord, and *none of the methods of treatment hitherto discussed has any influence over it*. Until its action is exhausted it continues to stimulate the motor and to a less degree the sensory tracts of the spinal cord, and kills the patient by exhaustion. The only way to remedy this state of affairs, so far as we know, is to depress the functions of the spinal cord. We have at our disposal a number of drugs whose main therapeutic action is to render the spinal cord less susceptible to stimulus. Administration of one or more of these remedies forms an integral part of any rational plan for the treatment of tetanus. The drugs most often employed are chloral, chloretone, and similar products; the bromides; physostigma, hyoscine, morphine, and magnesium sulphate.

The usual doses of these drugs, recommended for ordinary occasions, do not apply to the treatment of tetanus. The object is to give enough of the drug to produce therapeutic action, and until this has been attained the dose may be steadily increased. But there is one caution always to be kept in mind: this is, that the gastro-intestinal tract of a patient with tetanus may not absorb as readily as might be expected, and there is danger that drugs will accumulate in the bowels and be suddenly absorbed in overwhelming doses when a turn for the better occurs. This may be theoretical reasoning, but we are sure we have seen more deaths from tetanus with the patient completely relaxed than in convulsions. In our series of 23 cases (13 deaths) the condition at death is noted in all but 4 (Cases 1, 7, 17, 21); only 3 (Cases 4, 16, 18) died in spasm or convulsion, and 6 (Cases 2, 5, 12, 13, 19, 22) died in complete relaxation; and in some of these cases the condition was due to overaction of the spinal depressants employed.

We believe most reliance can be placed on the use of *chloral*

and the *bromides*. Hutchings employed *chloretone* in 6 cases with only 1 death, and he warmly advocates this drug. It is administered by mouth or rectum in doses of from 30 to 60 grains, dissolved in whisky or in hot olive oil. In our series it was employed three times: Case 12 died after being comatose for twenty-four hours; Case 13 died after being comatose for three hours; Case 20 died from puerperal sepsis. In both Cases 12 and 13 the administration of chloretone was stopped as soon as the patients began to show signs of coma, but without avail.

Morphine as the only depressant was used in only one case in this series (Case 21), the patient dying. We believe it is less effective as a spinal depressant than chloral and the bromides, and that it never should be employed to the exclusion of these drugs. In combination with other spinal depressants it was systematically employed only in Cases 1, 5, 22, and 23, three patients dying.

Hyoscine was used in Case 15, the patient recovering.

Chloral and bromides were employed in practically all cases except Case 21, in which morphine alone was used.

Magnesium Sulphate. In 1906, Blake adopted intraspinal (subdural) injections of magnesium sulphate in cases of tetanus. The treatment is based on the anæsthetic effect of intraspinal injections of this drug, as determined by Meltzer in 1905. A 25 per cent. watery solution of the chemically pure drug is employed, and 1 c.c. of the solution is used for every twenty-five pounds of body weight. In heavy adults, however, this dosage might prove excessive, as it might also in some women and young children. Fox, in 1910, collected 24 cases which had been treated by intraspinal injections of magnesium sulphate. Among these patients there were 11 deaths, a mortality of 45.8 per cent. Such isolated case reports have little value, as they give no true idea of the value of a remedy. Only when some large consecutive series of cases has been treated with magnesium sulphate can we determine its proper place in the treatment of tetanus.

According to Fox's table, J. N. Henry is the physician who has had most experience with this method of treatment. He reported 4 cases, with 3 deaths, 1 of which may have been caused by magnesium-sulphate poisoning. In our own series of cases intraspinal injections of magnesium sulphate were employed in 3 instances (Cases 17, 19, 22), and all the patients died, one (Case 19) undoubtedly of an overdose. Other cases have been reported in which death was attributed to the remedy rather than the disease (Henry, Hessert), or in which a fatal termination was averted solely by resort to active stimulation and artificial respiration (Soutter).

Magnesium sulphate has also been used subcutaneously. Miller quotes 3 mild cases with recovery, and Paterson reports 1 successful case.

The experiments of Camus, concerning the therapeutic value of magnesium sulphate have already been mentioned.

D. *The patient*, as well as the disease, must be treated; and we come finally to say a few words about feeding, nursing, etc. When first seen it is well to administer a purge; when the disease is once fully established, it may be very difficult to secure an evacuation of the bowels. Retention of urine must be watched for and relieved by the catheter. Isolation is desirable rather for the sake of protecting the patient from noise than for the purpose of preventing contamination of other patients, which is very rare.¹ Slamming doors, loud and especially sudden talking, and in fact noises of all kinds should be prevented. The patient's ears may be stopped with cotton, and the floor heavily carpeted. A bed that squeaks during the convulsions is a great annoyance. Window frames should be kept from rattling. Nursing must be con-

¹ Reynier says that in 1902 his assistant carried spores on his ungloved hands from one hospital, where he had amputated for tetanus, to another hospital where he infected 3 patients who died of tetanus after operations by this assistant. Now that gloves are habitually worn, such an occurrence could hardly take place.

stant and painstaking. Food must be administered at all hazards, by a nasal tube if necessary. Saline solution by the bowel, as in cases of peritonitis, tends to overcome the dehydration of the tissues produced by excessive muscular activity. Drugs may be administered in the rectal infusion, hypodermically, or by mouth.

SUMMARY. From the foregoing discussion it is evident that the rational treatment of tetanus comprises the five indications enumerated at p. 121. The *care of the wound*, both as a prophylactic and as a curative measure, is most important. The *neutralization of the toxin*, by the rational use of antitoxin, is indispensable; and we think we have demonstrated the inadequacy of the dosage usually employed for subcutaneous administration, and the necessity of intraneural, intraspinal, and probably also of intravenous injections. The excellent results reported in some quarters from the use of *carbolic-acid injections* should be remembered; it is a remedy much more readily obtained than antitoxin. The third indication, *to depress the functions of the spinal cord*, must not be met to the exclusion of the foregoing. Those who are enthusiastic in the use of intraspinal injections of magnesium sulphate seem to forget that unless they also employ antitoxin in a rational manner they are doing nothing to aid the body tissues to withstand the onslaught of the disease. Finally, the *care of the patient*, nursing and feeding, is the most practical part of the treatment, and one without which all the other parts may fail of their effect.

When we encounter another case of tetanus, we hope to be able to apply rational treatment in the following manner:

The patient will be placed in quiet, with competent nursing facilities. As soon as possible after coming under observation, whether this be in the small hours of the night or at bright noon-tide, the motor nerves leading from the wounded part will be exposed, as near to the cord as practicable, and as much antitoxin as each will contain will be

injected toward the spinal cord.¹ An intraspinal injection of at least 3000 units will then be made according to the usual technique for spinal anæsthesia. If it is possible to prick the cord with the needle, so much the better. Next the wound of entrance of the infection will be widely opened, all foreign bodies, sloughs, etc., will be removed by forceps, scissors, or scalpel; the wound will be irrigated with hot peroxide of hydrogen, swabbed out with 3 per cent. alcoholic solution of iodine, and loosely filled with gauze soaked in the same solution, and injection of antitoxin will be made (1500 to 3000 units) deeply into the muscular tissues around the wound. Continuous proctoclysis, as used in cases of peritonitis, will be given; and by mouth or in the rectal fluid will be administered effective doses of chloral and bromides, at appropriate intervals. Feeding will be enforced, by the nasal tube passed under chloroform anæsthesia, if necessary. During the course of the first day a moderate amount of antitoxin will be administered intravenously; probably 10,000 units will suffice.

The intraneural and intraspinal injections of antitoxin will be repeated daily, under chloroform anæsthesia, until marked decrease in spasticity occurs. Every twelve hours, or less often, a moderate amount of antitoxin will be injected intravenously, or even subcutaneously, so as to neutralize the circulating toxins; but the main reliance will be placed on intraneural and intraspinal injections. The administration of spinal depressants will be continued so long as they are indicated; a comatose state or muscular relaxation naturally

¹ For wounds of the *sole of the foot*, it is sufficient to inject the sciatic nerve; for those of other parts of the *lower extremity*, not alone the sciatic but the anterior crural and obturator nerves as well should be injected. For wounds of any part of the *upper extremity*, the brachial plexus should be exposed above the clavicle, and an injection should be made into each of its cords. These operations should be done under general anæsthesia, for which we prefer chloroform. A strong linen ligature is to be looped loosely around each of the nerves exposed; the ends of these ligatures are to be left long, and used to identify the nerves and draw them up into accessible positions for the purpose of subsequent injections of antitoxin should these prove necessary.

are contraindications. The wound will be dressed daily, as above described, until a healthy granulating surface is obtained.

With such treatment, commenced within twelve hours of the first appearance of symptoms of tetanus, we believe the mortality of the disease should not be over 20 per cent. Of the 11 patients under our own care, 7 have recovered and only 4 died, a mortality of 36.36 per cent. One of these deaths was caused by an overdose of magnesium sulphate; this patient did not come under observation until the fourth day of the disease, and none of the other fatal cases came under our care until more than twenty-four hours after the onset of indubitable symptoms of tetanus.¹

¹ Since the above was written, one of us (Ashhurst) has seen in consultation with Dr. George W. Norris, in his ward at the Pennsylvania Hospital, another case of tetanus:

Samuel W., negro, aged twenty-seven years, crushed his right index finger shortly before December 1, 1912. The skin was broken, but he bandaged the finger himself and never had any medical treatment for it. About December 8 or 9, he began to complain of stiffness and soreness in jaws, but never had any difficulty in swallowing. On December 11 he complained of a "ball of wind" in the epigastrium, which caused oppression and dyspnoea (tetanus of diaphragm?). Dyspnoea became more urgent on December 13, and on December 14 he went to bed. About this time he noticed that his legs were getting stiff (descending tetanus), and could be flexed only with great difficulty; no complaint of pain. Since December 15 he has had frequent spasms (ten to twelve daily) of his abdominal and thoracic muscles. These caused no pain, but made it more difficult to breathe. His legs were not weak, and would support him if he was helped up into a standing position; but they were perfectly helpless from rigidity.

Admitted to the Pennsylvania Hospital December 17, evening. Receiving ward diagnosis: *Transverse myelitis*. Transferred to medical ward and seen by Dr. Norris on the morning of December 18, when a diagnosis of *tetanus* was made.

First seen by Dr. Ashhurst at 6.45 P.M. on December 18. No antitetanic treatment had been instituted. During the day the patient had had five general convulsions, and had a sixth convulsion while under observation. When examined after this convulsion; there was no *trismus*; the head was retracted and the lumbar spine arched; the lower extremities were in full extension and very rigid. The patient lay on his right side.

December 18. At 7.30 P.M., under chloroform anæsthesia, lumbar puncture was done: there was a very free flow of spinal fluid, which was under great tension (5 c.c. were sent to the laboratory for examination

The following table gives the general mortality in some recent series of consecutive cases of tetanus:

(CONSECUTIVE) CASE REPORTS.

	Cases.	Recovered.	Died.	Mortality.
Bockenheimer (1908)	20	3	17	85.0 per cent.
Busch (1907)	30	9	21	70.0 per cent.
Episcopal Hospital (1905 to 1912)	23	10	13	56.5 per cent.
Fricker (1897 to 1902)	22	9	13	55.5 per cent.
Hessert (1909)	15	5	10	66.6 per cent.
Hofmann (1907)	30	20	10	33.3 per cent.
Huber (1912)	38	7	31	81.5 per cent.
Hutchings (1909)	6	4	2	33.3 per cent.
Jacobson and Pease (1906) . . .	191	58	133	69.6 per cent.
Kintzing (1911)	7	7	0	
Magula (1911).	33	11	21	66.7 per cent.
Simon (1911)	6	4	2	33.3 per cent.
Suter (1905)	14	2	12	85.7 per cent.

as to presence of tetanus toxin, but through some misunderstanding this examination was not made; a culture of the fluid remained sterile); 8000 units of antitoxin (15 c.c.) were injected into the subdural space of the cord; this was all the antitoxin available at that time. The patient was given chloral, grains xv, and potassium bromide, grains xxx, every three hours by mouth.

At midnight the patient was seen again, and 15,000 units of antitoxin were given intravenously in a pint and a half of saline solution. No more convulsions had occurred.

December 19. The next morning the patient was better. There had been no more convulsions, but many opisthotonic spasms. At 7.30 A.M. he was given a sponge bath, and after this he had two general convulsions. There was more trismus than at any time, and the tongue could just be protruded between the teeth. During the forenoon chloroform was again administered, and Dr. Norris attempted to give antitoxin intraspinally, but was unable to introduce the needle. Therefore 13,500 units were given intravenously in six ounces of saline solution. This made a total of 36,500 units administered in an effective manner within sixteen hours of the time the patient came under surgical observation. No intraneural injections were given, as no facilities existed for a surgical operation.

The patient was seen again at 7 P.M. There had been no convulsions all day. His muscles were quite relaxed. The chloral was reduced.

December 20. Has had no more convulsions; only a few, and not severe, spasms. Has taken food well. Chloral stopped.

December 21. Perfectly relaxed. *Cured of tetanus.* Clear in head and converses normally, but has pneumonia at right base (probably from inspiration of food) and is quite weak.

December 22. Died at night from pneumonia; temperature, 101° F., pulse, 148; respirations, 40.

CASES OF TETANUS AT EPISCOPAL HOSPITAL 1905 TO 1912.

Number.	Date of admission.	Age and sex.	Site of wound.	Date and nature of injury.	Incubation (time from injury to symptoms.)	Time from symptoms to institution of efficient treatment.	Type of case.	Summary of treatment.		Result.	Service of	Attending.
								Original injury.	Tetanus.			
1	Oct. 9, '05	23, M.	Left arm	Sept. 18, '05. Vaccination.	Unknown. Less than 19 days	3 days	Severe. Convulsions early.	Neglected.	Sedatives. Antitoxin, subcutaneously (60 c.c. = 18,000 units?) in 1 day.	Died, Oct. 10, '05	Sinkler	Sinkler and Sweeney.
2	June 4, '06	Boy	Left forearm	May 26, '06. Laceration from kick of horse.	9 days	12 hours	Severe. Convulsions early.	Ordinary antiseptic dressing.	Sedatives. Antitoxin, subcutaneously, and (3 hours before death) intraspinally (1500 units). Total amount 45,450 units in 2 days.	Died, June 6, '06	Neilson	Owen.
3	Aug. 6, '06	48, M.	Right hand	Aug. 6, '06. Compound fracture of wrist-joint. (Machinery crush.)	9 days	12 hours	Mild. No convulsions.	Ordinary antiseptic dressing.	Sedatives. Antitoxin, subcutaneously, 108,000 units in 2 days.	Recovered, Aug. 23, '06	Deaver	Weber and Aufhammer.
4	Mar. 5, '08	44, M.	Right toe	Jan. 9, '08. Cut by axe in felling a tree.	8 weeks	3 days	Severe. Convulsions early.	None for 5 weeks. Ordinary antiseptic dressing for 3 weeks.	Toe amputated. Antitoxin subcutaneously, 12,000 units in 1 day.	Died, Mar. 6, '08	Davis	Davis and Brown.
5	May 31, '08	26, F.	Left leg	May 31, '08. Compound fracture. Thrown into river by explosion.	Over 2 weeks	5 days, June 23, '08	Slow in onset. Convulsions late.	Ordinary antiseptic dressing.	Sedatives. Antitoxin, subcutaneously, 18,000 units in 5 days.	Died, June 30, '08. In coma 3 days before death	Neilson	Neilson and Price.

6	Aug. 12, '08	13, M.	Behind ear	Aug. 6, '08. Punctured wound by rusty knife.	4 days	2 days	Mild. No convulsions.	Puncturesutured; no drain.	Antitoxin subcutaneously, 3000 units in 2 days.	Recovered, Aug. 23, '08	Deaver	Deaver and Corson.
7	Aug. 28, '08	35, M.	Foot	Aug. 24, '08. Punctured wound by rusty nail.	5 days	1 hour	Severe. No convulsions.	No treatment for 3 days, then opened and drained.	Sedatives. Antitoxin, subcutaneously, 5000 units in 2 days.	Died, Aug. 30, '08	Deaver	Deaver and Corson.
8	Sept. 28, '08	34, M.	Abdomen and thigh	Sept. 24, '08. Brush burns, hematoma. Thrown from wagon and dragged.	14 days	1 hour	Slow in onset, but severe convulsions.	Ordinary antiseptic.	Sedatives. Antitoxin, subcutaneously, (129,000 units) and intraspinally (3000 units). Total antitoxin 132,000 units in 10 days.	Recovered Oct. 31, '08	Frazier	Ashhurst and Corson.
9	Sept. 23, '08	55, M.	Cranium	Sept. 23, '08. Gunshot fracture of temporal region and rupture of eye-ball.	4 weeks	12 hours	Mild. Slow in onset. No convulsions.	Aseptic. No operation.	Sedatives. Antitoxin, subcutaneously, (31,500 units in 8 days).	Recovered. Eyeball enucleated, Nov. 17, '08	Frazier	Ashhurst and Aufhammer.
10	Nov. 12, '08	49, M.	Foot	Nov. 4, '08. Punctured wound by rusty nail.	8 days	24 hours	Medium severity. No convulsions.	Home treatment, by ham fat, etc.	Sedatives. Antitoxin, intraspinally (3000 units), and later subcutaneously (27,000 units). Total amount, 30,000 units in 6 days.	Recovered, Nov. 18, '08	Frazier	Ashhurst and Gracey.
11	Dec. 19, '08	19, M.	Foot	Nov. 30, '08. Punctured wound by splinters from mill floor.	18 days	24 hours	Severe. Convulsion before admission.	Wound dressed twice but patient never returned for further dressings.	Three splinters removed from wound. Sedatives. Antitoxin intraspinally, and intraneurally, and into muscles around wound. Total in 2 days 4500 units.	Recovered, Dec. 31, '08	Frazier	Ashhurst and Gracey.
12	Oct. 16, '09	63, M.	Fingers	Sept. 28, '09. Lacerations by machinery.	18 days	24 hours	Severe. No convulsions.	Ordinary antiseptic dressings.	Fingers amputated. Sedatives (chlore-tone). Antitoxin intraspinally, 1500 units.	Died, Oct. 19, '09 After being comatose for 24 hours	Frazier	Ashhurst and Hopper.

Number.	Date of admission.	Age and sex.	Site of wound.	Date and nature of injury.	Incubation (time from injury to symptoms).	Time from symptoms to institution of efficient treatment.	Type of cases.	Summary of treatment.		Result.	Service of	Attending.
								Original injury.	Tetanus.			
13	Nov. 20, '09	11, F.	Knee	Nov. 11, '09. Abrasion from fall.	9 days	40 hours	Slow in onset. Severe. Convulsions late.	Bread-poultice at home.	Sedatives (chlore-tone). Antitoxin, intraneurally, 3 times (total 9000 units), intraspinally (1500 units), subcutaneously (3500 units). Total antitoxin 13,500 units in 3 days.	Died, Nov. 22, '09 after being comatose for 3 hours	Frazier	Ashhurst and Hopper.
14	Feb. 4, '10	45, F.	Toes	Jan. 23, '10. Laceration. Fall down stairs in bare feet.	12 days	Few hours	Mild. Slow onset. No convulsions.	Neglected for 10 days. Then iodine.	Sedatives. Antitoxin, subcutaneously, 6000 units in 3 days.	Recovered, Feb. 19, '10	Morris	Ashhurst and Siner.
15	Feb. 21, '10	23, F.	Scalp	Jan. 26, '10. Lacerated wound. Hit by brick.	19 days	1 week	Slow in onset. Severe. No convulsions.	Sutured, no drain.	Abscess opened. Sedatives. Antitoxin, intraspinally, 3000 units; subcutaneously, 21,500 units. Total antitoxin 24,500 units in 5 days.	Recovered, Mar. 8, '10	Neilson	Ashhurst and Siner.
16	July 17, '10	8, M.	Thumb	July 5, '10. Cut by hatchet.	10 days	2 days	Slow in onset. Severe. Convulsions.	Sutured, no drain.	Sedatives. Antitoxin, subcutaneously, 103,000 units in 3 days.	Died, July 20, '10 in spastic state	Deaver	Griffith
17	Oct. 14, '10	7, M.	Arm	Sept. 24, '10. Vaccination.	Unknown. Less than 3 weeks	12 hours	Mild. Slow in onset. No convulsions.	Neglected.	Sedatives, and magn. sulph. intraspinally. Antitoxin subcutaneously, 60,000 units in 2 days.	Died, Oct. 16, '10	Frazier	Frazier and Henneberger.

18	Oct.	3, '10	6, F.	Arm	Sept. 7, '10.	Vaccination.	Unknown Less than 24 days	3 days	Slow onset. Very severe. 2 convulsions every hour.	Treated at home by mother.	Sedatives. Antitoxin, intraspinaly, 10,000 units; subcutan- eously, 45,000 units. Total antitoxin, 55,- 000 units in 2 days.	Died, Oct. 5, '10 in convulsion	Frazier	Ashhurst and Griffith.
19	Feb.	4, '11	6, M.	Hand	Dec. 23, '10.	Laceration. Fall on icy street.	9 days	4 days	Severe. Convulsions.	Sutured.	Sedatives, and magn. sulph. intraspinaly. Antitoxin, intra- spinaly, 9500 units; subcutaneously, 10,000 units. Total antitoxin, 19,500 units in 2 days.	Died, Jan. 7, '11 in perfect relaxation	Frazier	Ashhurst and Johnston.
20	Feb.	4, '11	27, F.	Uterus	Jan. 9, '11.	Instrumental delivery.	Less than 22 days	4 days	Mild onset. Chronic. No convulsions.	Uterus packed for secondary hemorrhage Jan. 19, '11.	Sedatives, chloretone Phenol, 3 per cent. sol. for 1 day. Anti- toxin, subcutan- eously 26,500 units in 10 days.	Recovered, but died 9 days later (Feb. 22, '11) of puer- peral sepsis	Mutschler	Mutschler and Johnston.
21	Apr.	17, '11	18, M.	Fingers	Apr. 10, '11.	Crush by machine.	7 days	Few hours	Severe. No convulsions.	Fingers ampu- tated.	Abscesses drained. Sedative, morphine only. Antitoxin sub- cutaneously, 30,000 units in 4 days.	Died, Apr. 19, '11	Neilson	Alexander and Campbell.
22	Oct.	3, '11	18, M.	Scrotum	Sept. 23, '11.	Punctured wound 3 inches deep from fall on rusty iron.	10 days	Few hours	Very severe. Convulsions.	No treatment.	Sedatives, and magn. sulph. intraspinaly. Antitoxin subcuta- neously, 59,000 units in 4 days.	Died, Oct. 7, '11	Frazier	MacFarland
23	Mar.	15, '12	28, M.	Foot	Mar. 5, '12.	Punctured wound by brass bolt.	8 days	2 days	Slow onset. Severe. No convulsions.	Bandaged. No antiseptic treat- ment.	Sedatives. Antitoxin, subcutaneously, 224,000 units in 3 days.	Recovered, Apr. 3, '12	Neilson	John.

ANALYSIS OF EPISCOPAL HOSPITAL CASES

Twenty-three cases. Mortality, 56.5 per cent.

10 recovered (3, 6, 8, 9, 10, 11, 14, 15, 20, 23).

13 died (1, 2, 4, 5, 7, 12, 13, 16, 17, 18, 19, 21, 22).

Incubation under ten days, 11 cases. Mortality, 63.6 per cent.

4 recovered (3, 6, 10, 23).

7 died (2, 7, 13, 16, 19, 21, 22).

Incubation over ten days, 12 cases. Mortality, 50 per cent.

6 recovered (8, 9, 11, 14, 15, 20).

6 died (1, 4, 5, 12, 17, 18).

Efficient treatment within twelve hours of symptoms, 5 cases. Mortality, 20 per cent.

4 recovered (9, 10, mild cases; 11, 23, severe cases).

1 died (17, severe case).

No efficient treatment within twelve hours of symptoms, 18 cases. Mortality, 66.6 per cent.

6 recovered (3, 6, 8, 14, 15, 20, all mild cases).

12 died (1, 2, 4, 7, 12, 13, 16, 18, 19, 21, 22, severe cases; 5, mild case).

Less severe type of disease, 9 cases. Mortality, 11 per cent.

8 recovered (3, 6, 8, 9, 10, 14, 15, 20).

1 died (5).

Very severe type of disease, 14 cases. Mortality, 85.7 per cent.

2 recovered (11, 23).

12 died (1, 2, 4, 7, 12, 13, 16, 17, 18, 19, 21, 22).

Wounds of upper extremity, 9 cases. Mortality, 89 per cent.

Wounds of lower extremity, 8 cases. Mortality, 50 per cent.

Wounds of trunk, 3 cases. Mortality, 33.3 per cent.

Wounds of head, 3 cases. Mortality, 0.0 per cent.

Antitoxin used in all 23 cases:

Efficiently as to method and quantity in 12 cases.

Mortality, 46.1 per cent.

7 recovered (3, 8, 9, 10, 11, 15, 23).

5 died (13, 16, 17, 18, 19).

Inefficiently in 11 cases. Mortality, 72.7 per cent.

3 recovered (6, 14, 20).

8 died (1, 2, 4, 5, 7, 12, 21, 22).

CASE HISTORIES

CASE 1.—Edward C., aged twenty-three years. Admitted, October 9, 1905. Discharged, October 10, 1905. Died. Service of Dr. Sinkler. Attending, Drs. Sinkler and Sweeney.

Left arm was vaccinated September 18; it began to get sore in four days, and patient had slight nausea. Then felt well until October 7 (nineteen days after vaccination), when he had headache, pain in back of neck, nausea, and vomiting, with beginning trismus. Family physician dressed arm and advised removal to hospital, but patient stayed home two days longer.

On admission (third day after first symptoms) there was trismus and retraction of head, abdomen scaphoid and rigid. On arm a slough the size of a silver dollar, baring muscle, which is black and sloughing and surrounded by area of necrotic fat; no pus.

October 9. On admission, given morph. sulph., gr. $\frac{1}{4}$, at 9 A.M., 4 P.M., and 9 P.M.; tetanus antitoxin, 20 c.c. in morning, repeated in afternoon, hypodermically. At 6 P.M. there was suggestion of a convulsion.

October 10. Convulsion at 6 A.M. for four minutes. Legs rigid, slight opisthotonos; spasms at approach of anyone. Given potass. bromide, gr. xl, and chloral hydrate, gr. xxx, by enema; also morph. sulph. at 3 A.M. and 4 A.M., hypodermically. Also 20 c.c. of antitoxin. *Died*, 8 A.M.

CASE 2.—George V., schoolboy (age unknown), under twelve years. Admitted, June 4, 1906. Discharged, June 6, 1906. Died. Service of Dr. Neilson. Attending, Dr. Owen.

On May 26, was kicked by a horse, sustaining green-stick fracture of left forearm, with lacerations of skin; not a compound fracture. Treated in surgical dispensary for the next week. On June 2 felt ill, went to bed, but was up and about the next day. On June 4 complained of *bad pain in affected arm*, trismus and

headache; had one or two slight convulsions, and was sent to the hospital.

On admission (same day as first symptoms). Jaws could be opened one-half inch; neck was stiff, abdomen rigid, legs stiff, but not rigid. Knee-jerks present on both sides, also ankle clonus. Is able to talk, has no pain until disturbed, then becomes temporarily rigid.

June 4. On admission, given bromide, gr. xv, and chloral hydrate, gr. v, every four hours by rectum. Given 30 c.c. of tetanus antitoxin every three or four hours, hypodermically.

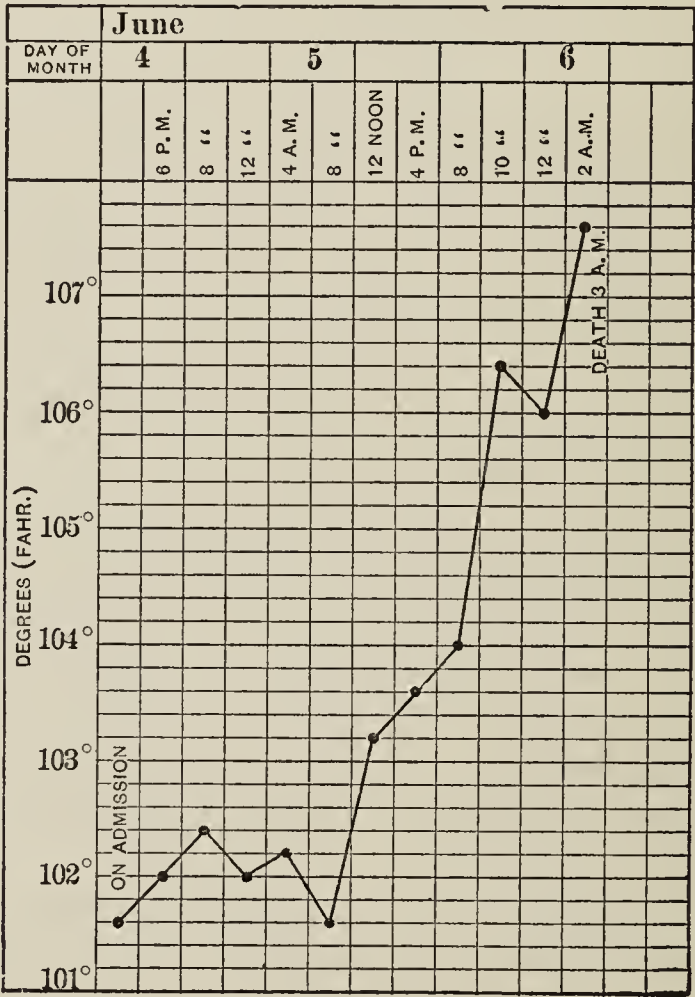


FIG. 42.—Typical temperature chart of fatal case. (Case 2.)

June 5. Dose of tetanus antitoxin increased to 60 c.c. every four hours hypodermically. Given in all 330 c.c. of antitoxin hypodermically.

June 6. Given 10 c.c. of antitoxin (1500 units) intraspinally, at 12.30 A.M. Died at 3.15 A.M., “evidently from sudden paralysis of respiratory centres.”

CASE 3.—Steven C., aged forty-eight years. Admitted, August 6, 1906. Discharged, September 2, 1906. Recovered. Service of Dr. Deaver. Attending, Drs. Weber and Aufhammer.

Right hand caught in machinery on morning of admission; diagnosis on admission, compound fracture of wrist-joint, badly lacerated and contused. Wound did fairly well.

August 15. (Nine days after injury.) Developed trismus and some stiffness of muscles of neck.

August 16. Given sodium bromide, gr. x, and chloral hydrate, gr. x, every six hours; also tetanus antitoxin, 30 c.c. every two hours hypodermically.

August 17. Given sodium bromide, gr. xv, and chloral, gr. lx, every six hours; also antitoxin as before.

August 19. Given sodium bromide, gr. xx, and chloral, gr. x, every six hours.

August 20. Sodium bromide, gr. xv, and chloral, gr. x, every six hours. Same treatment continued until

August 23. When patient developed delirium tremens, pneumonia, and later empyema. Transferred to surgical ward. *Cured of tetanus*, September 2, 1906. *Died* after many months as result of empyema.

CASE 4.—James McD., aged forty-four years. Admitted, March 5, 1908. Discharged, March 6, 1908. Service of Dr. Davis. Attending, Drs. Davis and Brown. Died.

Eight weeks ago, while chopping down a tree, patient lacerated his right great toe with an axe. Came to surgical dispensary five weeks later (three weeks ago). On March 2, began to have pain in back; pain and stiffness increased.

March 3. Neck and jaw became stiff. This morning the jaw became fixed. No convulsions. Walked to hospital.

On admission. Jaws can be opened one-quarter of an inch; muscles stiff and spastic. Dr. Davis amputated the toe under ether. Patient had a convulsion during the operation.

Antitoxin, 3000 units, at 6 P.M., subcutaneously; antitoxin, 3000 units, at 10.30 P.M., subcutaneously.

March 6. Antitoxin, 3000 units, at 4 A.M., subcutaneously; antitoxin, 3000 units, at 9 A.M., subcutaneously. At 10 A.M. the patient *died of asphyxia in convulsions*.

CASE 5.—Katie M., aged twenty-six years. Admitted, May 31, 1908, at 12.30 A.M. Discharged, June 30, 1908, 5.35 P.M. Died. Service of Dr. Neilson. Attending, Drs. Neilson and Price.

Admitted for compound fracture of left tibia and fibula, simple fracture of right tibia and fibula. Injured by explosion in naphtha launch on the Delaware River, all sixteen passengers being thrown into the river. Patient badly shocked.

June 20. For some days has had slight spasticity of facial muscles and tendency to sardonic grin, more noticeable in the morning; some stiffness of muscles at back of neck; no convulsions. Sodium bromide, gr. xx, and chloral, gr. v, every four hours.

June 21. Jaws, back, and neck still stiff; facial muscles, better.

June 22. Trismus continues, but is no worse; swallows easily; says she bites her tongue whenever she sleeps.

June 23. Patient not so well; jaws stiffer; twitches more; difficulty in swallowing; neck stiff and painful. Antitoxin, 3000 units, twice (6000 in all, two hours apart). Sodium bromide, gr. xx, and chloral hydrate, gr. x, every four hours. Morph. sulph., gr. $\frac{1}{8}$, and atropine sulphate, gr. $\frac{1}{150}$, every six hours.

June 24. Antitoxin, 3000, t. i. d. (9000). Has a slight general convulsion each time disturbed.

June 25. Temperature, pulse, and respiration, rising. Temperature, 106.4° F.; comatose; sedatives stopped; sponged, p. r. n.

June 26. Conscious and restless; sedatives again.

June 27. Less restless. Antitoxin, 3000 units, at 5.30 P.M.

June 28. Comatose; Cheyne-Stokes' respiration.

June 29. Comatose; did not move all day.

June 30. Comatose; *died* at 5.35 P.M.

CASE 6.—Leslie R. E., aged thirteen years. Admitted, August 12, 1908. Discharged, August, 23 1908. Recovered. Service of Dr. Deaver. Attending, Dr. Corson.

Six days before admission was struck behind the right ear with a rusty knife, which stuck in his neck; this penetrated about one and one-half inches, and was withdrawn with difficulty; small wound (punctured by thin blade); taken to another hospital where suture was put in wound and no attempt at drainage made. Began to feel stiffness at back of neck, headache and fever, and twitching of face muscles four days later (two days before admission).

On admission. Small crust over wound behind right ear, with one silkworm gut suture; this was removed, wound opened and packed. Antitoxin, 1500 units, hypodermically.

August 13. Still slight stiffness of jaws. Antitoxin, 1500 units, hypodermically.

August 14. All muscular stiffness gone.

August 20. Out of bed.

August 23. Discharged. Diagnosis of tetanus considered positive at the time in the opinion of Dr. George Thomas (family physician) and Dr. Deaver.

November 17, 1912. Returned for examination. Has had no symptoms since. In perfect health. Small cicatrix, scarcely visible, over base of right mastoid process.

CASE 7.—Andrew H., aged thirty-five years. Admitted, August 28, 1908. Discharged, August 30, 1908. Died. Service of Dr. Deaver. Attending, Drs. Deaver and Corson.

Ran a rusty nail into his foot on August 24; came to the dispensary on August 27, because foot swelled up. Wound was opened "clear through," but is still painful, and is kept in the ward because he lives at a distance.

August 29. At 4 P.M. the patient suddenly developed stiffness of jaws and back of neck. Very much excited and irritable. Foot laid freely open where the nail had penetrated. Carbolized and packed. Antitoxin, 2000 units, subcutaneously. Sodium bromide, gr. xx, and chloral, gr. x, every six hours.

August 30. More opisthotonos; very restless. Antitoxin, 3000 units, hypodermically. *Died* at 10 P.M.

CASE 8.—Joseph G., aged thirty-four years. Admitted, September 28, 1908. Discharged, November 3, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Corson.

Four days before admission was thrown from a wagon and was dragged, receiving a brush burn of the abdomen. Referred to ward as recent accident, because of hematoma of the left thigh.

October 6. Hematoma incised.

October 7. Jaws stiff. Antitoxin, 1500 units, subcutaneously.

October 8. Jaws still stiff; no pain except when he eats; a little twitching at night.

October 9. Stiffness in abdomen and across lower back. Jaws can be opened one-half inch. Headache all night. Antitoxin, 1500 units. Transferred to isolation ward.

October 10. At 2.30 A.M. had convulsion, tetanic in type. Threw his head back and had opisthotonos. Given chloroform and 4 c.c. of slightly turbid cerebrospinal fluid withdrawn and 3000 units of antitoxin injected. Brush burn curetted. Sodium bromide, gr. xx, and chloral, gr. xx, every six hours; 9 A.M., patient rather delirious; a little nauseated; back of neck stiff. Antitoxin 3000 units, every four hours, subcutaneously. (Stopped October 17.) 10 A.M., jaws open one-quarter inch; some pain in head.

October 12. Headache ceased.

October 13. Abdomen much less rigid. Neck limber and back much less rigid.

October 14. Jaws open one inch.

October 17. Antitoxin reduced to 1500 units, every four hours. Sedatives decreased.

October 21. Slightly comatose.

October 24. Abdomen soft.

October 31. Sent back to ward as *recovered*.

CASE 9.—Thomas W., aged fifty-five years. Admitted, September 23, 1908. Discharged, December 5, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Aufhammer.

A gunshot wound of the right temporal region, with a fracture of the orbit and rupture of the eyeball. Dr. G. O. Ring in consultation for the ocular condition.

October 20. Four weeks after injury. Complains of stiffness of jaws, says it started October 19; 1500 units antitoxin, hypodermically. Sodium bromide, gr. xx, every six hours. Transferred to isolation ward.

October 21. Antitoxin, 1500 units, every four hours.

October 23. Antitoxin, 1500 units, every eight hours.

October 24. Antitoxin, 1500 units, every ten hours.

October 26. Antitoxin, 1500 units, every twelve hours.

October 27. Antitoxin, 1500 units, every sixteen hours. *Cured* of tetanus, October 27, and transferred to convalescent ward, where Dr. Ring enucleated eyeball on November 17.

CASE 10.—John S., aged forty-nine years. Admitted, November 12, 1908. Discharged, December 8, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Gracey.

Chief complaint. Stiffness of jaws and sore foot.

November 4. The patient ran a rusty nail through the shoe into his foot. Pulled the nail out and washed the foot with chloride of lime and soda ash and put ham fat on it. The foot began to swell, and he used iodine and arnica. One week after injury to the foot jaws began to stiffen so that he could not eat. Came to the dispensary and was sent to the ward November 12.

On Admission. Jaws can be opened one-half inch; right leg is rigid and painful. Has had spasms of jaws, which snap shut and cannot be opened.

November 12. At 7 P.M., under chloroform, 3000 units of tetanus antitoxin was injected into the subdural space of the cord; the foot, which meanwhile had healed, was reopened through the plantar fascia widely, and 1500 units of antitoxin injected deeply into muscles of foot; 1500 units of antitoxin injected, every four hours, hypodermically for five doses.

Summary (until morning of November 14). First day: Antitoxin intraspinally, 3000 units; into wound, 1500 units; subcutaneously, 7500 units. Total 12,000 units. Calomel, gr. ss; mag. sulph., oz. j; whisky, fl. oz. iij; chloral, gr. lx; potassium bromide, gr. cxx.

November 14 and 15. Second day: Antitoxin, 13,500 units, subcutaneously; chloral, gr. lx; potassium bromide, gr. clx; whisky, fl. oz. vii; calomel, gr. j; mag. sulph., oz. j.

November 15. Third day: Antitoxin, 4500 units, subcutaneously; potassium bromide, gr. lxxx; whisky, fl. oz. vss.

November 16. Fourth day: No antitoxin. No sedatives. Patient transferred to men's medical ward for bronchitis.

November 26. Patient given 3000 units of antitoxin hypodermically for pain in jaws. No further symptoms of tetanus.

November 17, 1912. Returns for examination, four years after recovery. Says for two years right foot and leg troubled him, being sometimes weak, dragging as he walked, but at other times had no trouble. In December, 1910, on stepping suddenly off a trolley car, right leg became spastic, jaws locked, and patient had to grasp a street post to prevent falling. Under great muscular effort he managed after several minutes to open his mouth. No such attack since; but occasionally had shooting pains in leg and up the spine to the head. Has been under medical treatment for the past year, and has had no trouble with his leg during that time. Until the last year has had to be laid off work for several weeks at a time once or twice annually. Physical examination is negative. Linear, supple scar on sole of right foot, two inches long. Knee-jerks normal. No paresis or spasticity.

CASE 11.—Thomas C., aged nineteen years. Admitted, December 12, 1908. Discharged, December 31, 1908. Recovered. Service of Dr. Frazier. Attending, Drs. Ashhurst and Gracey.

November 30. While running along floor of mill, soaked in machine oil, where he works, a large splinter ran through a hole in his shoe and penetrated the sole of his left foot near the head of the metatarsal of the great toe. The patient went to the dispensary, where the wound was cauterized and drained. Second visit to the dispensary two days later, but made no further visits, the patient himself removing catgut drainage that had been introduced. Was away from his work for one week.

December 17. The eighteenth day after the injury. Patient did not feel well and his jaws were sore and stiff. Took to his bed on December 18. That night he says his back muscles con-

tracted until he rested only on the back of his head and heels. Was sent to the hospital by family physician who saw him in the morning.

On Admission. Jaws can be opened one-half inch; considerable stiffness of muscles of the back of neck and some stiffness of the back muscles; abdomen markedly rigid and a tendency to stiffness of the legs. Wound on foot has counter-opening for drainage, two inches distant. As soon as possible after admission patient was given chloroform and 3000 units of antitoxin injected intraspinally; the wound in the foot was opened deeply through the plantar fascia, scraped out and packed with gauze soaked in tincture of iodine, 1 part to 3 of water. Three splinters removed from the wound. Culture on blood serum and an anaërobic culture made (streptococci; no *Bacillus tetani*).

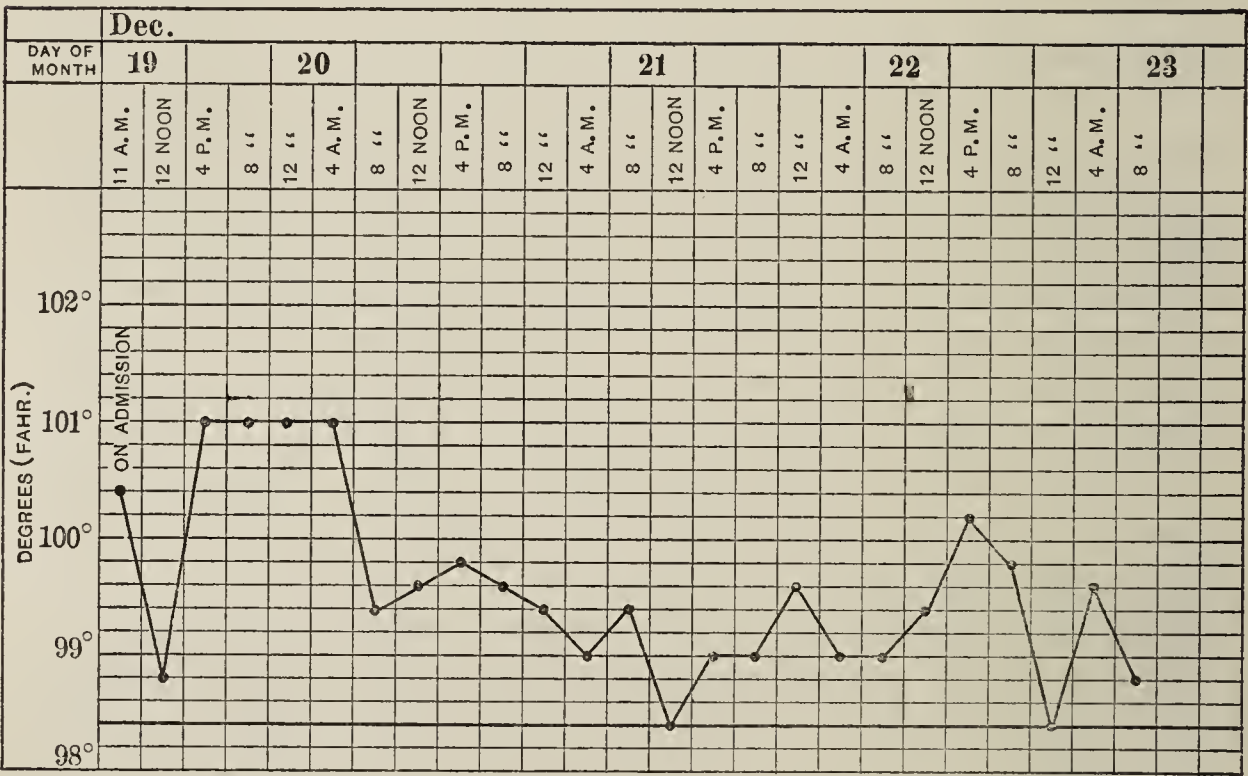


FIG. 43.—Typical temperature chart of severe case arrested promptly with ultimate recovery. (Case II.)

December 20. Jaws opened one and one-eighth inches; not so stiff in the neck muscles. Chloral hydrate, gr. x, and potassium bromide, gr. xx, every three hours. Total chloral hydrate, gr. lx, and potassium bromide, gr. cxx. At 3.30 P.M., 750 units of anti-toxin was injected into the sciatic nerve, which was exposed by an incision under chloroform, and 750 units injected around the wound in the foot.

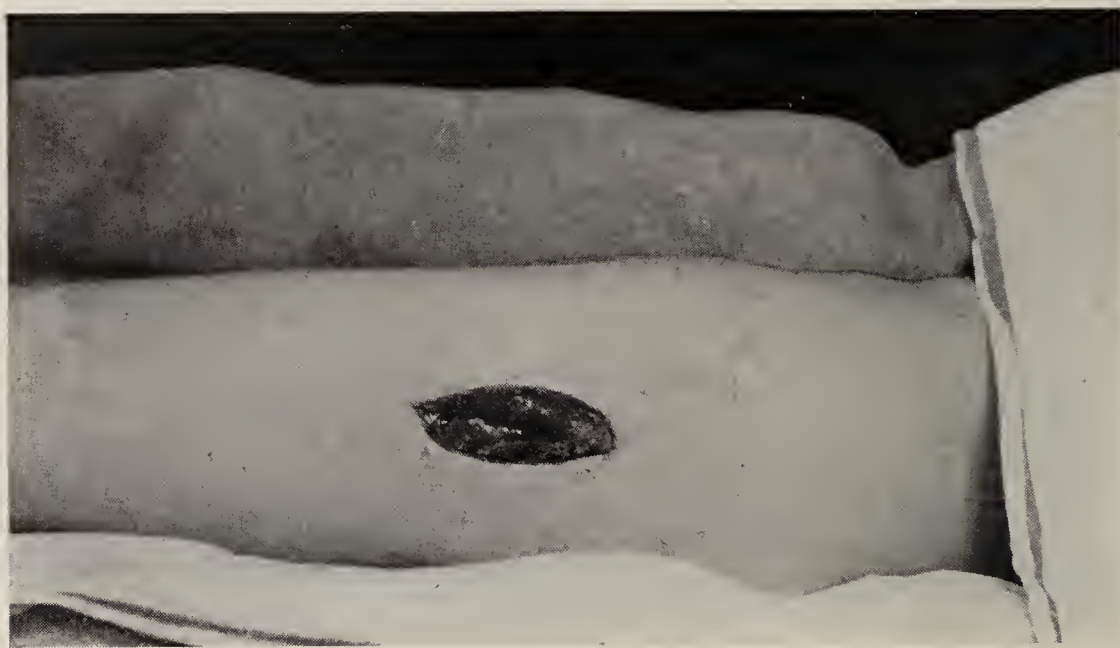


FIG. 44.—Granulating wound through which antitoxin had been injected into the sciatic nerve. Wound left open for subsequent injections, which, however, were not necessary. (Case II.)

December 21. Jaws opened one and three-quarter inches. Summary: No antitoxin; chloral hydrate, gr. c; potassium bromide, gr. clx.

December 22. Summary: No antitoxin; chloral hydrate, gr. lxxx; potassium bromide, gr. clx.

December 23. Summary: No antitoxin; chloral hydrate, gr. l; potassium bromide, gr. c.

December 24. Summary: No antitoxin; chloral, gr. xxxv; potassium bromide, gr. lxx.

December 25. Summary: No antitoxin; chloral, gr. xx; potassium bromide, gr. l. Patient is now convalescent. Chloral and bromides continued in diminishing doses for three days longer.

December 31. Patient transferred to surgical ward as *cured* of tetanus.

CASE 12.—Francis C., aged sixty-three years. Admitted, October 16, 1909. Discharged, October 19, 1909. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Hopper.

September 28. Fingers of the right hand badly lacerated in a picker in a woollen mill. Patient treated in the dispensary and attending daily.

October 15. Patient felt jaws becoming stiff and sent for the ambulance twelve hours later.

On Admission. October 16. General physique poor; jaws stiff; opisthotonos; abdominal muscles board-like; neck retracted; reflexes, especially the patellar, markedly increased. Chloral, gr. x, and sodium bromide, gr. xx, every two hours.

October 17. General condition poor. Under chloroform lumbar puncture was made and 1500 units of antitoxin injected. All the fingers of the right hand amputated at the metacarpal joint; even under chloroform the marked lordosis could not be made to disappear. Chloretone, gr. xxx, every two hours. Stopped the sodium bromide and chloral.

October 18. Patient semicomatose; heart bad. P.M. Heart weakening. No increase of rigidity.

October 19. Patient *died* at 4 A.M.

CASE 13.—Margaret D., aged eleven years. Admitted, November 20, 1909. Discharged, November 22, 1909. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Hopper.

November 11. Patient fell and abraded right knee. Was treated at home and became infected. Jaws became stiff in the morning of November 19, but went to school and on return again called mother's attention to her stiff jaws. Went to bed and slept

well. In the morning (November 20) jaws were rigidly clenched, and when she tried to move or be moved, she became very rigid; she had pain in jaws, neck, and back. Her family physician saw her, said she had symptoms of lockjaw, left some medicine and asked to be called on the phone in the evening. When he heard her condition then he sent a note asking for the ambulance and her admission to the hospital.

On Admission, November 20, 10.30 P.M., jaws set, head retracted, legs extended and rigid; moderate lordosis; board-like scaphoid abdomen; mind clear; reflexes all increased; suppurative abrasion over tubercle of the right tibia covered with a bread poultice.

November 21, 12.30 A.M. (two hours after admission). Patient was operated upon. (Chloroform.) The anterior crural and obturator and sciatic nerves were exposed and 750 units of antitoxin injected into each of anterior crural and obturator and 1500 units into sciatic; about 1500 units injected around wound in the leg, after disinfecting it and swabbing it out with 3 per cent. alcoholic solution of iodine. Continuous saline proctoclysis. Chloretone, gr. xv, every four hours by mouth. Chloral hydrate, gr. xx; and sodium bromide, gr. xxx, every two hours by rectum in saline proctoclysis. During the morning (November 21) the patient was quiet, jaws slightly less rigid and can now open one-quarter of an inch. Seems better generally.

2.30 P.M. Incisions reopened and same dose of antitoxin injected into anterior crural, obturator, and sciatic nerves; 1500 units of antitoxin given intraspinally under chloroform.

4.30 P.M. Teeth can be separated further.

7.30 P.M. Patient not so well. Temperature, pulse, and respiration all rising.

10.30 P.M. Patient restless. Jaws still less rigid. Clonic contractions of muscles of back and legs every few minutes; 1500 units of antitoxin hypodermically.

November 22, 1.30 A.M. No change.

9.30 A.M. Patient weaker. Chloretone stopped. Tincture of digitalis, minims v. Camphorated oil, minims xx, every four hours.

12.00 M. Antitoxin as above into nerves under chloroform; none given intraspinally.

3.00 P.M. Patient has been comatose ever since chloroform this noon. Respirations shallow and labored. Pulse better.

6.30 P.M. Patient *died*, in relaxation.

CASE 14.—Sarah McV., aged forty-five years. Admitted, February 4, 1910. Discharged, February 19, 1910. Recovered. Service of Dr. E. J. Morris. Attending, Drs. Ashhurst and Siner.

January 23 (twelve days before admission). When pregnant three months, fell down stairs in bare feet and sprained back and received small lacerated wound between fourth and fifth right toes; no other injuries. The interdigital cleft became very painful the next day and the foot began to swell. Patient treated the foot at home for ten days and then on February 2, came to the surgical dispensary and was dressed. Returned two days later and complained of pain on opening mouth and stiffness of jaws. Referred at once to isolation by Dr. Ashhurst, after renewed antiseptic treatment of wound with iodine (3 per cent.).

On Admission, February 4. Chief complaint: Stiffness of jaws and neck and wound of right foot.

Examination. Reflexes slightly increased; no ankle clonus or Babinski; abdomen not rigid; patient three months pregnant; 3000 units of antitoxin subcutaneously on admission. Potassium bromide, gr. xxx, and chloral hydrate, gr. xv, every four hours. Mag. sulph., oz. ss.

February 5. Patient complains of headache, backache, and muscular pains all over; abdomen somewhat tender and rigid. Reflexes slightly increased; 1500 units of antitoxin, one-half around wound in foot and one-half in abdominal wall, hypodermically. Bromides and chloral only t. i. d.

February 6. Pains in pelvis; uterine bleeding all day, passing several large clots.

Pelvic examination: Large soft cervix; fundus above pubis; os not dilated.

February 7. At 2 A.M. a very profuse metrorrhagia. Os now patulous and placenta protruding. Uterus cleaned out with placental forceps and packed. Fluid extract of viburnum prunifolium, drachm j every three hours.

10.00 A.M. Patient in good condition.

February 8. Packs removed; 1500 units of antitoxin subcutaneously.

February 19. Patient has done well. Discharged today as *cured* of tetanus.

CASE 15—Mary S., negro, aged twenty-three years. Admitted, February 21, 1910. Discharged, March 8, 1910. Recovered. Service of Dr. Neilson. Attending, Drs. Ashhurst and Siner.

January 26, at 2 A.M., while walking on the street, the patient was struck on the head by a brick, causing a slight laceration. She was taken in the ambulance to another hospital, where the laceration was sutured and the patient discharged one hour later. Returned on the tenth day and had the sutures removed. She says that at that time there was a small tender swelling in the wound.

February 14. One week before admission, and nineteen days after the wound, the patient's jaws gradually became painful, and she began to have difficulty in opening her mouth. The condition gradually grew worse, and she can now barely separate her jaws one-quarter of an inch, with much pain.

On Admission, February 21. Temperature, 100° F. Chief complaint is stiffness of jaws. Postauricular lymph nodes on right enlarged. Cicatrix from recent wound on scalp, nearinion; below is a small, tender lump. Abdomen a little tender. Some rigidity of muscles of back and neck. Patellar reflexes greatly exaggerated on the right and increased on the left. No ankle clonus or Babinski. Mag. sulph., oz. ss., stat. Antitoxin, 3000 units subcutaneously on admission and 1500 units intraspinally. Potassium bromide, gr. xxx, and chloral hydrate, gr. vii, every three hours by mouth. Hyoscine, gr. $\frac{1}{100}$, hypodermically, 3 P.M. and 11 P.M. Abscess on scalp opened.

February 22. Antitoxin 3000 units subcutaneously at 10 A.M. Sedatives as before. Patient's condition not so good. Jaws nearly closed. Neck and back somewhat rigid. Stuporous, and mind wanders. More pain in head. Antitoxin, 3000 units, at 4 P.M., subcutaneously; 1500 units at 8 P.M., when there was some improvement. Jaws not so rigid.

February 23, 10 A.M., 2 P.M., and 8 P.M., 1500 units of antitoxin subcutaneously. Condition the same. Morphine, gr. $\frac{1}{8}$. Great pain, worse in the back. Sedatives as before.

February 24, A.M., and P.M., 1500 units of antitoxin subcutaneously. Jaws opened one-half inch in the morning, and open one inch by evening. Sedatives as before.

February 25. No more sedatives after February 24; 1500 units of antitoxin subcutaneously. Tincture of digitalis, minims x, t. i. d.

February 26, 3000 units of antitoxin subcutaneously. Pains in lower jaw.

February 27. No antitoxin after this date.

February 28. Patient can flex neck until chin is within two inches of chest.



FIG. 45.—Opisthotonos. Death six hours later in convulsions. (Case 18.)

March 1. Can make chin touch chest.

March 2. Sat up in bed for first time. Soft diet for first time.

March 3. In chair for one-half hour.

March 4. Jaws open three-eighths of an inch.

March 5. In chair for two hours.

March 6. Patient walked a little.

March 7. Jaws can be opened one-half inch. Neck still somewhat rigid, and when it is flexed until chin touches sternum she has some pain in the back.

March 8. Discharged as *cured*.

CASE 16.—Charles M., aged eight years. Admitted, July 17, 1910. Discharged, July 20, 1910. Died. Service of Dr. Deaver. Attending, Dr. Griffith.

July 5. Was cut on right thumb by hatchet. Wound was dressed in dispensary, using three sutures. No symptoms until Friday evening, July 15, when he complained of a little difficulty in swallowing. On July 16, felt all right. On July 17, before admission, had a spasm which lasted only a few minutes.

On Admission. Patient perfectly relaxed, with the exception of some slight stiffness of jaws, and a sardonic grin, which was marked. Given 43,000 units of antitoxin hypodermically. Chloral, gr. ij, and potassium bromide, gr. vi, by mouth, every three hours. Had three spasms on day of admission, each lasting about three minutes, after which he was relaxed.

July 18. Temperature higher. General condition not so good. Better toward night. Five spasms during the day, each about three minutes, one severe. Opisthotonos marked in last convulsions and relaxation afterward not so complete. Sweating is marked; 5000 units of antitoxin, every three hours, for eight doses—40,000 units, hypodermically. Bromide and chloral as before.

July 19. General condition good, but jaws more locked. Some difficulty in swallowing. Pain in abdomen. Cyanosis of lips marked during spasms. Four spasms today, one quite severe; 20,000 units of antitoxin.

July 20. General condition not so good. Jaws locked. Took a bad turn at 9 P.M. Temperature, 106° F. *Died* at 10.30 P.M.

CASE 17.—Harry F., aged seven years. Admitted, October 14, 1910. Discharged, October 16, 1910. Died. Service of Dr. Frazier. Attending, Dr. Henneberger.

Three weeks ago the patient was vaccinated, and was all right until the evening of October 13, when malaise was noted and he

complained of stiffness of back and soreness in jaws. Brought to the hospital the next morning.

Examination. Jaws somewhat stiff and unable to open mouth to full extent. Abdomen scaphoid, with great rigidity of abdominal muscles. The least tapping throws them into a tetanic contraction; 5000 units of antitoxin, every four hours, subcutaneously. Mag. sulph. (intraspinally), 2 c.c. of 25 per cent. solution at 12.30 A.M., October 15.

October 16. *Died* at 1 A.M. Temperature, 104° F. Total amount of antitoxin in two days, 60,000 units subcutaneously.

CASE 18.—Minnie P., aged six years. Admitted, October 3, 1910. Discharged, October 5, 1910. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Griffith.

September 7. Patient was vaccinated. She did well. Scab formed, which was knocked off at play and no other scab formed. Mother dressed the wound and a slight infection occurred, but healing proceeded normally from outside toward the centre. No untoward occurrence until October 1, when the child complained of mouth being sore, but was able to eat and play and slept well the night of October 1. Complained of mouth all of October 2, but could swallow, and went to Sunday school. On Monday, October 3, had malaise, and a weak spell in which she fell and received an ugly contusion on forehead. During the day she still complained of mouth being sore and some slight stiffness. General muscular rigidity noticed, and jaws were somewhat hard to open. Brought to the hospital on the night of October 3.

On Admission. Contusion on the forehead the size of an orange, received by a fall in a weak spell. Opisthotonos; sardonic grin marked; angles of mouth drawn down; muscular rigidity marked; complains of pain in stomach; head retracted; had one slight convulsion before admission; sent to isolation ward, October 4; 5000 units of antitoxin subcutaneously on admission.

October 4. Muscular rigidity marked; opisthotonos and sardonic grin marked. Patient able to swallow. Had about two convulsions an hour today, but very slight. Potassium bromide, gr. lx, and chloral, gr. xxx, every three hours; 25,000 units of antitoxin subcutaneously.

October 5. Seen by Dr. Ashhurst on this date first; 10,000 units of antitoxin intraspinally, and 15,000 units of antitoxin subcutaneously. Temperature rising. Convulsions increasing in severity. Patient is very restless, but able to take nourishment and medicine. A very severe convulsion at 3 P.M., followed by

two slighter ones. Temperature, 106° F. At 4 P.M. several slight convulsions; breathing very labored; cold, clammy sweat. At 5.30 P.M. had three very severe convulsions, and *died* at 5.45 P.M. Temperature, 106.6° F.

CASE 19.—Thomas B., aged six years. Admitted, January 4, 1911. Discharged, January 7, 1911. Died. Service of Dr. Frazier. Attending, Drs. Ashhurst and Johnston.

Patient has a tapeworm, and has lost weight in the last few months. On December 23, while running in the street, he fell over a Belgian block, striking the palm of the right hand against a sharp piece of ice. A U-shaped laceration at the thenar eminence resulted, which bled very profusely. Was taken to another hospital, where two sutures were inserted, which controlled the hemorrhage. No antitoxin was given.

January 1. It was first noticed that his jaw was stiff and that he spoke like a child who was tongue-tied. He was taken back to the same hospital, where, on account of the difficulty in opening the mouth and some patches on the tongue, diphtheria was suspected, but by mouth gag no membrane was found in the throat.

January 4. A positive diagnosis of tetanus was made, and he was sent to the Episcopal Hospital.

On Admission. Teeth cannot be separated more than one-half inch. Face is wrinkled and angles of the mouth are drawn down and out in a sardonic grin. Depressors of the jaw become tense on any attempt to open mouth. When the patient is quiet, nothing abnormal is apparent. Neck muscles and those of the back are a little stiff, but no opisthotonos is present. Limbs are normal. A clean granulating wound on the right thenar eminence. 12 midnight; 5000 units of antitoxin in right forearm.

January 5, 4.00 A.M. Chloral hydrate, gr. iiij.

6.00 A.M. Slight convulsion.

8.00 A.M. Jaws more rigid.

9.00 A.M. 5000 units of antitoxin intraspinally.

1.30 P.M. Potassium bromide, gr. v. Weaker. Delirious. Temperature, 104° F.

4.00 P.M. Chloral hydrate, gr. iiij. Patient sleeping.

8.00 P.M. 5000 units of antitoxin subcutaneously. Potassium bromide, gr. v.

12.00 P.M. Chloral, gr. iiij. Temperature, 103.4° F.

January 6, 4.00 A.M. Potassium bromide, gr. v. Temperature, 100.2° F.

12.00 M. 4500 units of antitoxin intraspinally. Seen by Dr. Ashhurst on this date first.

1.00 P.M. Potassium bromide, gr. v. Temperature, 101.8° F. Chloral, gr. iiss by rectum.

4.00 P.M. Sleeping quietly.

8.00 P.M. Potassium bromide, gr. v, and chloral, gr. iiss, by rectum, which was expelled.

9.00 P.M. Potassium bromide, gr. v, and chloretone, gr. v.

11.00 P.M. Sleeping.

January 7. 1.00 A.M. Chloretone, gr. v, because he awoke.

3.00 A.M. Potassium bromide, gr. v. Temperature, 100.4° F.

9.00 A.M. Potassium bromide, gr. v.

12.00 M. Temperature, 101° F.

2.00 P.M. Mag. sulph., $3\frac{3}{4}$ grams in a 25 per cent. solution intraspinaly (7 c.c. of solution, clearly an overdose).

3.00 P.M. Patient *died* in perfect relaxation. Probably would have recovered if no magnesium sulphate had been given.

CASE 20.—Lillie S., aged twenty-seven years. Admitted, February 4, 1911. Discharged, February 22, 1911. Recovered. Service of Dr. Mutschler. Attending, Drs. Mutschler and Johnston.

Personal History. Has had one child. A prolonged and difficult labor with a laceration of the cervix. During the present gestation health has been good. Much better than previously.

Present Condition. January 9. While at breakfast, had a sudden profuse hemorrhage. The uterus was evacuated by her physician. Improved for ten days, when a second sudden very severe hemorrhage occurred. Two days later developed phlebitis in the left leg. Twenty-two days after delivery, four days before admission, patient began to have dull pains in muscles at back of neck, some difficulty in separating jaws, and slight pain on deglutition; all symptoms increased slowly in severity daily. On the day before admission had 5000 units of antitoxin subcutaneously.

On Admission. Able to open incisor teeth only enough to admit tip of the index finger. This causes some pain in the neck. Some rigidity of muscles of back of neck, and pain on motion. Uterus felt just above pubis.

Vaginal Examination. Small amount of brownish discharge, not especially foul. Cervix soft and lacerated; uterus enlarged, soft, not freely movable. Left leg uniformly increased in size; skin glistening, pits slightly on pressure. No pain in leg or on pressure over veins.

February 4, 6.00 P.M. Chloretone, gr. x, every four hours.

8.00 P.M. 5000 units of antitoxin in pectoral muscle. Left leg bandaged.

February 5, 8.00 A.M. Good bowel movement; appetite good; considerable thirst. Unable to open jaws enough to admit spoon.

4.00 P.M. Phenol, 3 per cent., minims xv, every two hours, hypodermically.

8.00 P.M. Antitoxin, 5000 units, in right pectoral muscle. Patient very drowsy. Pain less in the neck.

February 6, A.M. Patient very drowsy.

4.00 P.M. Temperature, 104.8° F.; pulse, 136; respiration, 28.

5.00 P.M. All treatment stopped. Atropine sulphate, gr. $\frac{1}{100}$; morph. sulph., gr. $\frac{1}{5}$, hypodermically.

8.00 P.M. All appearances of intense sepsis. Completely relaxed. No pain anywhere. Infusion of digitalis, fl. dr. ij. Enteroclysis with normal salt solution, to take one pint every three hours; with each pint, two drachms infusion of digitalis.

February 7, 12.00 M. Antitoxin, 5000 units, subcutaneously.

Return of pain and stiffness. Chloretone, gr. x, every three hours.

February 10. 5000 units of antitoxin subcutaneously.

February 11. 5000 units of antitoxin.

February 13. 1500 units of antitoxin.

February 22. Patient gradually improved until morning of February 22, when all tetanic symptoms had been absent for some days. On latter date, she had a severe uterine hemorrhage. Uterus packed, but patient failed rapidly.

February 23, 12.15 A.M. Patient *died*.

CASE 21.—Poland P., aged eighteen years. Admitted, April 17, 1911. Discharged, April 19, 1911. Died. Service of Dr. Neilson. Attending, Drs. Alexander and Campbell.

April 10. While at work the patient had his hand caught in a machine; index, ring, and middle fingers cut. Middle and ring fingers amputated on the same day. Discharged on April 14. Returned to hospital on April 17, with tetanus. Jaws locked, pain in back, and stiffening of neck muscles. Hand is very much swollen, and has a very offensive odor. Stitches removed from fingers. Palm of hand opened up and one fluidounce of pus removed. Several stab wounds made on back of hand; 5000 units of antitoxin given subcutaneously every seventh hour. Hand dressed every day. Continuous 1 to 5000 bichloride dressing on hand.

April 17. Antitoxin, 10,000 units, subcutaneously; whisky, oz. j, and morph. sulph., gr. $\frac{1}{4}$.

April 18, A.M. Antitoxin, 5000 units, subcutaneously; whisky, oz. jss, and morph. sulph., gr. $\frac{1}{4}$. P.M. Antitoxin, 10,000 units, subcutaneously; whisky, oz. ij, and morph. sulph., gr. $\frac{1}{4}$.

April 19. Patient *died* at 3.55 A.M.

CASE 22.—John McD., aged eighteen years. Admitted, October 3, 1911. Discharged, October 7, 1911. Died. Service of Dr. Frazier. Attending, Dr. MacFarland.

September 20. The patient fell on a rusty hydrant stalk; the stem entered the scrotum, and he walked home untreated. Later on the same day he came to the surgical dispensary, where he was not treated, but referred to ward, but did not stay. No antitoxin given. Thirteen days later was admitted to the hospital with symptoms of tetanus. (How long these had existed is not stated; probably two days.)

On Admission. Well-developed adult male. Temperature, 99° F.; pulse and respiration, normal. Has a sardonic grin; head retracted; slight opisthotonos; occasional attacks of general rigidity; jaws can be opened one-half inch; over pubis in the midline is an area the size of a quarter dollar, which is tender, red, and inflamed; open wound on left side of scrotum, discharging yellow, purulent material. Probe reaches from here to area above pubis under the skin.

Treatment. Under local anæsthesia, incision made over tender area at pubis, and through-and-through drainage to scrotal wound with rubber tube. Antitoxin, 14,000 units in 5000 doses subcutaneously. Potassium bromide, drachm ij, and urotropin, gr. x.

October 4. Patient is worse. Frequent attacks of spasms, and pulse and respiration have increased. Antitoxin given every six hours, to make 20,000 units, subcutaneously. Frequent convulsions, with opisthotonos lasting two to three minutes. Potassium bromide, drachms ix; urotropin, gr. x; morph. sulph., gr. $\frac{1}{4}$.

October 5. Potassium bromide, drachms ivss, and urotropin, gr. v. Antitoxin, 10,000 units, subcutaneously. Morph. sulph., gr. $\frac{3}{4}$; intraspinal injection of mag. sulph., 25 per cent. solution, 6 c.c.

October 6. Patient has developed pneumonia, and is in a critical condition. No convulsion since early morning. Antitoxin, 15,000 units, subcutaneously. Potassium bromide, drams iiss; urotropin, gr. xv; atropine, gr. $\frac{1}{75}$; mag. sulph., intraspinally, 3 c.c. of a 25 per cent. solution; whisky, oz. iiiss; tincture of digitalis, minims xl, hypodermically, in 10-minim doses.

October 7. Patient in very critical condition. *Died* at 6 P.M.

CASE 23.—Gregol S., aged twenty-eight years. Admitted, March 15, 1912. Discharged, April 20, 1912. Cured. Service of Dr. Neilson. Attending, Dr. John.

Chief complaint is rigidity of jaw and neck muscles; wound of right foot.

March 5. The patient ran a piece of a brass bolt into the outer side of his foot, and went to a "lodge doctor," who bandaged it without any further treatment. Eight days later, on Wednesday, March 13, began to have pain and rigidity of muscles of jaw and neck. Was admitted on Friday night, March 15, with well-marked trismus and considerable rigidity and retraction of neck with moderate arching of the back.

On Admission. Symptoms as above. Face is drawn, mouth particularly being drawn at the corners into the typical tetanic risus sardonicus. The head is rigid and well drawn back. Complains of some pain in the neck and thorax. Is able to separate the teeth about one inch.

Thorax: Lungs negative. Heart area normal and sounds good. No murmurs. Back arched from occiput to buttocks, but the legs are freely movable. Centre of arch about three inches above the bed.

Abdomen: Board-like rigidity of all the abdominal muscles, increased on examination. A small inguinal hernia on right side.

Extremities: All are normal, with the exception of the right leg, which shows a penetrating wound on the outer side of the right foot about the middle of the arch. This runs up and back under the skin below the external malleolus for about three inches.

March 15. Wound in foot opened its whole length; slough cut away; washed with peroxide and warm boric, and packed with iodoform gauze; 6000 units tetanus antitoxin injected subcutaneously. Chloral hydrate, gr. xx, and potassium bromide, gr. x, every four hours, with morph. sulph., gr. $\frac{1}{8}$, hypodermically, every four hours.

March 16. 93,000 units of antitoxin injected subcutaneously. Patient is not so well. Can barely separate teeth, and has convulsive spasms every few seconds. No real convulsion, however.

March 17. 65,000 units of antitoxin injected subcutaneously. Patient can open mouth a little wider, but the convulsive spasms keep up, and there is more rigidity and arching of the back.

March 18. 60,000 units of antitoxin injected subcutaneously. Patient in about the same condition. No worse.

March 19. No antitoxin. Patient has had first real sleep this morning. Slept again for several hours in the afternoon. Convulsive spasms are not quite so frequent, nor so severe. Arching and rigidity not diminished, possibly slightly increased.

March 24. Patient is much improved, and rests quietly. Bromide and chloral reduced by half.

March 26. Patient rests quietly, but rigidity continues undiminished, and considerable urticaria over body. Given soft-boiled egg and a small piece of dry toast for dinner.

March 27. Rigidity considerably decreased, patient in good condition.

March 30. Patient's general condition is good. Rigidity is well marked but decreasing slowly; most marked in abdominal muscles. Blood pressure of left arm: systolic, 182; diastolic, 124.

March 31. Hot bath for fifteen minutes seemed to decrease rigidity to a considerable extent. Returned later in the day, but the patient was much more comfortable.

April 3. Transferred to Convalescent Ward.

April 7. Patient is doing well, and is up and in chair daily.

April 16. Patient put on full diet. Is up and walking about the ward. Is still a little weak, and is slightly sore in the chest, but on the whole is in good condition.

April 20. Patient is up and about. Still has slight soreness over lower thorax. Discharged in good condition.

November 17. Returned for examination. No symptoms since recovery, and in excellent health now.

REFERENCES

- Almagia and Mendes. *Internat. Clinics*, Philadelphia, 1908, iii, 12.
 Bacelli. *London Med. Mag.*, 1893-'94, ii, 811.
 Beates and Thomas. *Monthly Cyclop. and Med. Bull.*, 1911, iv, 326.
 Blake. *Surg., Gyn., and Obst.*, 1906, i, 541.
 Bockenheimer. *Arch. f. klin. Chir.*, 1908, lxxxvi, 277.
 Busch. *Arch. f. klin. Chir.*, 1907, lxxxii, 27.
 Camus. *C.-R. Soc. de Biol.*, Paris, 1912, lxxii, 109.
 Dehne and Hamburger. *Wien. klin. Woch.*, 1904, xvii, 807.
 Donitz. *Deutsch. med. Woch.*, 1897, xxiii, 428.
 Fedden. *Clin. Jour.*, 1911, xxxvii, 356, Case 2.
 Fink. *Jour. of Trop. Med.*, 1911, xiv, 161.
 Fox. *New York Med. Rec.*, 1910, ii, 720.
 Frazier. *Keen's Surgery*, Philadelphia, 1906, i, 478.
 Fricker. *Deutsch. Zeit. f. Chir.*, 1907, lxxxviii, 429.

- v. Graff. *Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1912, xxv, 145.
Gumprecht. *Deutsch. med. Woch.*, 1894, xx, 546.
Gumprecht. *Arch. f. d. ges. Physiol.*, 1894, lix, 105.
Hessert. *Surg., Gyn., and Obst.*, 1909, ix, 145.
Hitchens. *American Vet. Rev.*, August, 1910.
Hofmann. *Beitr. z. klin. Chir.*, 1907, lv, 697.
Huber. *Beitr. z. klin. Chir.*, 1912, lxxvii, 139.
Hutchings. *Surg., Gyn., and Obst.*, 1909, ii, 11.
Imperiali. *Giorn. di Med. Milit.*, 1910, fasc. x to xi: in *Policlinico*, 1911, xviii, 363.
Jacob. Blumenthal and Jacob: *Berl. klin. Woch.*, 1898, xxxv, 1079.
Jacobson and Pease. *Trans. Amer. Surg. Assoc.*, 1906, xxiv, 254.
Kintzing. *New York Med. Jour.*, 1911, xi, 1268.
Krauss and Amiradzibi. *Zeit. f. Immunitätsforsch.*, 1912, vii, 1.
Küster. *Centr. f. Chir.*, 1905, xxxiii, Beil., S. 15.
v. Leyden. *Berl. klin. Woch.*, 1899, xxxvi, 632; *Deut. med. Woch.*, 1901, xxvii, 477.
Lop. *Bull. Soc. Chir., Paris*, 1906, xxxii, 184.
Luckett. *Med. and Surg. Repts., Bellevue and Allied Hosps., New York*, 1904, i, 319.
Magula. *Beitr. z. klin. Chir.*, 1911, lxxvi, 588.
Maunoury. *Assoc. Franç. de Chir.*, 1902, xv, 608.
Marie and Morax. *Ann. de l'Inst. Pasteur*, 1902, xvi, 818; 1903, xvii, 335.
Matas. *Trans. Amer. Surg. Assoc.*, 1909, xxvii, 40.
McCambell. *Jour. Amer. Med. Assoc.*, 1907, i, 919.
McFarland. *Jour. Amer. Med. Assoc.*, 1903, ii, 34.
Meyer and Ransom. *Arch. f. exp. Pathol. u. Pharm.*, 1903, xlix, 369.
Miller. *Amer. Jour. Med. Sci.*, 1908, ii, 781.
Neary. *New York State Jour. of Med.*, 1910, x, 476.
Nissen. *Deutsch. med. Woch.*, 1891, xvii, 775.
Nocard. *Bull. Acad. de Méd., Paris*, 1895, xxxiv, 407.
Palmer. *Jour. Royal Army Med. Corps*, 1912, xviii, 400.
Paterson. *Lancet*, 1910, i, 922.
Porter and Richardson. *Boston Med. and Surg. Jour.*, 1909, clxi, 927.
Pribram. *Prag. med. Woch.*, 1908, xxxiii, 719.
Remertz. *Inaug. Dissert., Berlin*, 1911. Ueber prophyl. Injektion. von Tetanus Antitoxin.
Reynier. *Bull. Acad. de Méd., Paris*, 1908, lix, 623, 629, 780.
Riche. *Bull. et Mém. Soc. Chir., Paris*, 1912, xxxviii, 476.
Rogers. *Jour. Amer. Med. Assoc.*, 1905, ii, 12.
Roux and Borrell. *Ann. de l'Inst. Pasteur*, 1898, xii, 225.
Sawamura. *Arbeiten a. d. Inst. z. Erforschung d. Infektionskrankh. in Bern (Kolle)*, Heft iv, 1, Jena, 1909.
Schnitzler. *Centr. f. Bakteriolog.*, 1893, xiii, 679.
Sewaki. *Sei-i-Kwai Med. Jour.*, Tokyo, 1910, xxx, 33.

- Simon. v. Graff: *Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1912, xxv, 145.
Solieri. *Centr. f. Bakteriolog.*, 1910, lv, 141.
Suter. *Arch. f. klin. Chir.*, 1905, lxxv, 113.
Stintzing. *Münch. med. Woch.*, 1898, xlv, 1265.
Stintzing. *Mitth. a. d. Grenzgeb. d. Med. u. Chir.*, 1898, iii, 461.
Tarozzi. Cited. by Solieri, q. v.
Vaillard. *Bull. Acad. de Méd., Paris*, 1908, lix, 569, 587.
Vincent. *Ann. de l'Inst. Pasteur*, 1904, xviii, 748; *Soc. de Biol. de Paris*, in *Gaz. des Hôp.*, 1908, lxxxi, 620, 668, 921.
Zupnik. *Deutsch. med. Woch.*, 1900, xxvi, 837.

THE CLINICAL SIGNIFICANCE OF EXTREME DEGREES OF HIGH BLOOD PRESSURE, WITH REMARKS ON ITS MANAGEMENT¹

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I WOULD be tempted to ask your indulgence for speaking to you on so commonplace a topic as high blood pressure, were it not for the frequency with which this phenomenon is encountered and the diagnostic and prognostic difficulties which this symptom presents. At the outset it should be clearly understood that hypertension is in no sense a clinical entity; like fever it is but a symptom, the expression of some morbid process, hence its recognition entails the necessity of searching for the underlying cause.

For the most part cases with high blood pressure fall into one of three classes: (1) Chronic nephritis, especially the chronic interstitial type; (2) arteriosclerosis; and (3) a group of cases in which neither of these conditions is present to account for the elevation in blood pressure. This group has been designated simple high tension of unknown origin. This last group corresponds with the cases emphasized by Janeway and more recently by Riesman, in which without demonstrable vascular lesions or noteworthy urinary findings a persistent arterial hypertension exists. As pointed out by Osler, it is well-nigh impossible in these cases to exclude alterations in the splanchnic vessels or other internal vascular areas. Ultimately it will doubtless be shown that these cases do depend upon localized vascular disease.

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1. CHRONIC NEPHRITIS. Clinically nephritis is found to be the most frequent cause of extreme hypertension. The variety of nephritis associated with the most marked increase of blood pressure is the chronic interstitial type. High blood pressure also occurs in chronic parenchymatous nephritis, but it is found in only about one-half of the cases, and according to Buttemann its extent depends upon the amount of interstitial change that has taken place in the renal tissue. It has of late been conclusively demonstrated that even in acute nephritis elevation of blood pressure takes place, and Gordon found that in acute nephritis in children this rise of pressure was often very great. Acute scarlatinal nephritis is particularly prone to cause hypertension, and instances are on record in which within twenty-four hours of the onset of such an acute nephritis a decided rise in pressure occurred. This fact goes to show that in the hypertension of kidney disease the renal lesion is primary and that the cardiovascular lesions, the arteriosclerosis and hypertrophied heart, are secondary phenomena.

Bright himself recognized the frequency with which increased arterial tension accompanies nephritis and since his observations much able experimentation has been carried out in the effort to demonstrate the underlying cause of the high blood pressure in kidney disease.

Bright considered that some abnormal condition of the blood either stimulated the heart to overaction or so affected the arteries and capillaries that increased resistance was offered to the efforts of the heart to drive the blood through the vessels. Later G. Johnson advanced the idea that owing to deficient renal elimination the blood contained abnormal constituents, which through the vasomotor nerves so acted on the arteries that they were stimulated to contract, as a result of which increased resistance occurred in the vessels, which in turn caused the heart to hypertrophy. About the same time Traube advanced the purely mechanical theory for the cardiac hypertrophy and hypertension in nephritis.

He held that the diminution in the vascular area of the kidney introduced a resistance to the circulation and that in the attempt to overcome the obstacle the heart underwent hypertrophy and the blood pressure was raised. Cohnheim upheld this hypothesis in a modified form. Abundant experimental proof to the contrary has shown this mechanical view to be untenable, since it has been repeatedly demonstrated that ligation of both renal arteries, or ligation of the abdominal aorta, immediately above the renal vessels fails to cause hypertension. Furthermore, Alwens has shown that when the kidneys are subjected to an external pressure sufficient to block completely the flow of blood through these organs, no significant rise of blood pressure results. Pässler and Heineke were not convinced that the rise of pressure and cardiac hypertrophy which they found in dogs after excising increasing quantities of kidney tissue was due entirely to the diminution in the capillary area of the kidneys. Janeway also found hypertension and cardiac hypertrophy in dogs after causing a reduction of the renal substance by ligation of several branches of the renal arteries. But Pearce in his Harvey Lecture, which furnishes an admirable critical survey of the entire subject, points out that in all these reduction experiments the procedures themselves are such as to produce, in addition to the loss of substance, atrophic and vascular renal changes accompanied by the elimination of albumin and casts, so that in reality a true chronic nephritis is produced, and the observation furnishes no proof that restriction of the vascular area of the kidney *per se* is responsible for the production of hypertension.

Gull and Sutton maintained that general arteriocardiac fibrosis was always the primary disturbance responsible on the one hand for the cardiac hypertrophy, and on the other for the renal lesion. This supposition is more than likely true in the so-called arteriosclerotic kidney, but is inapplicable to other forms.

The theory of Ewald that the cardiac hypertrophy was due

to increased viscosity of the blood has been disproved by the accurate observations of Hirsh and Beck, who showed that no definite relationship exists between the viscosity of the blood and cardiac hypertrophy with hypertension.

Innumerable other conflicting theories have been postulated, but, in 1905, Loeb, a pupil of Krehl, put forth an explanation for the hypertension of renal diseases which has been accepted by not a few. Loeb found that hypertension occurs chiefly in those cases of nephritis in which there is extensive disease of the glomeruli. The activity of the kidneys is dependent upon the amount of blood flowing through the glomeruli at a given time. Loeb holds that when owing to increased capillary resistance within the glomeruli local vaso-dilatation is inadequate to meet the functional demands of the kidneys, an impulse is conveyed to the central nervous system which induces reflexly splanchnic vaso-constriction in order that the general blood pressure may be raised and an adequate rate of blood-flow may be maintained through the contracted glomeruli.

In contradistinction to these theories, of late the more purely chemical theories are gaining in favor. Bingel and others have shown that there exists in the kidneys a pressor substance capable of raising blood pressure, although Pearce failed to demonstrate this substance in extracts of dogs' kidneys. It is assumed by some that in nephritis a pressor substance circulates in the blood, thereby causing vascular spasm. Whether this substance is newly formed in the diseased kidneys or whether there is only an increase in quantity and liberation of a normally present pressor material, is a matter for conjecture.

Others maintain that the vascular phenomena of nephritis are the result of the presence of adrenalin in the blood; notably Schur and Weisel, who assert that they have demonstrated the presence of adrenalin in the blood of nephritics, and attribute the existence of hypertension to this substance. They further describe in these cases of chronic renal disease

constant hypertrophic changes in the medulla of the adrenal gland. Aschoff, Cohn, and Goldschmidt, on the other hand, have failed to find this hypertrophy of the adrenal medullary structures and hold that the tests used by Schur and Weisel, to demonstrate adrenalin in the blood, are not specific for that substance.

It will be seen from this review of the more important hypotheses as to the cause of hypertension and cardiac hypertrophy in nephritis, that as yet no definite conclusions can be drawn and that we are still far from arriving at the truth of the matter. On the whole, the chemical theory would appear to be the more attractive and is capable of being applied to the majority of cases. Nevertheless, regardless of their ultimate cause, the existence of increased vascular tension and cardiac hypertrophy as concomitant signs of chronic nephritis is established beyond peradventure. Furthermore, it should be noted that observers are generally agreed that these cardiovascular phenomena in nephritis are of a compensatory character and represent an effort on the part of nature to maintain at a level somewhere near normal the functional activity of the most important excretory organs of the body.

Before leaving this subject of hypertension in nephritis attention should be directed to the fact that when in the course of chronic renal disease low blood pressure exists, it is generally evidence of circulatory failure and usually the well known symptoms of decompensation soon supervene. There are, however, cases of albuminuria occurring usually in young adults which are constantly associated with normal or low blood pressures and which show no abnormal cardiac signs. It is doubtful whether these cases should be regarded as true nephritis.

2. ARTERIOSCLEROSIS. High blood pressure does not occur as frequently as the result of arteriosclerosis alone as it does of nephritis. The explanation of this lies in the fact that arteriosclerosis may be local or general and whether or not

blood pressure is elevated depends largely upon the distribution of the sclerosis. It has frequently been shown that local arteriosclerosis, especially if it involves only the peripheral vessels, may be intense and yet be unaccompanied by any noteworthy increase in blood pressure. This is readily seen in senile patients with pipe stem radials, whose blood pressure is virtually normal.

Noteworthy hypertension occurs in arteriosclerosis practically only when the process involves the splanchnic vessels or the intrathoracic aorta. Experimental proof of this is the fact that, after eliminating all vasoconstrictor reflexes by section of the cervical cord, ligation of the abdominal aorta will raise blood pressure only when the ligature is placed above the origin of the coeliac axis. This accounts for the frequent occurrence of exceedingly high blood pressures in those patients whose peripheral vessels are free of sclerosis, so far as may be judged by palpation. When we consider what a large proportion of the vascular tree is embraced in the splanchnic area it is not surprising that partial obliteration of these vessels should have an effect on the general arterial tension. Why sclerosis of the thoracic aorta should act in a like manner is more difficult of comprehension. Bittorf ingeniously advances the hypothesis that degeneration of the pressor nerve or its ganglia in the aortic arch is responsible for the high pressure under these circumstances. The pressor nerve conveys impulses to the brain which send out in response vasodilator impulses, thereby lowering arterial tension. Degeneration of this nerve or its terminations obviously would destroy the possibility of this mechanism for vasodilatation.

The hypertension of arteriosclerosis may occur under two conditions: When it occurs early in the disease it is the result of spasm of the vessels and when it appears later it is due to the secondary fibroid and calcareous changes within the vessel walls which cause loss of elasticity and finally obliteration of the lumen. From the prognostic standpoint this

difference is of the utmost importance, since in the first instance the hypertension is amenable to treatment, whereas in the second little can be done to alter the condition. In most developed cases there is usually an element of spasm present along with the fibrosis.

When cases of arteriosclerosis exist in which ordinarily there is no elevation of blood pressure demonstrable, in spite of probable visceral sclerosis, the explanation may be two-fold: In the earlier stage of the disease the lumen of the vessels is not encroached upon and the elasticity of the vessel walls is still sufficient to meet the requirements of an orderly, quiet existence without causing increased arterial tension. When, however, such a case is subjected even to moderately increased physical or mental exertion the vessel walls are too inelastic to accommodate themselves to the increased blood-flow and as a result undue tension occurs in the vessels. This increase of tension is manifest not so much by an abnormal increase of the systolic pressure, but rather by a disproportionate elevation of the diastolic and consequent diminution of pulse pressure, since it is at this time in the cardiac cycle that the degree of vascular tension is especially conspicuous.

The second cause for lack of blood pressure elevation in arteriosclerosis is to be found in the nutritive changes which occur as a result of sclerosis especially of the coronary arteries. Coronary sclerosis leads to faulty nutrition of the cardiac musculature and consequent myocardial degeneration, which results in weakened cardiac power and hence a lowering of blood pressure. In this instance the lowering is chiefly at the expense of the systolic pressure and the diastolic may remain abnormally high. This may readily account for the low systolic blood pressure noted in those older people with marked peripheral sclerosis, since it is altogether probable that in these patients considerable degeneration or even atrophy of the cardiac musculature may have taken place.

Our conception of the blood pressure in arteriosclerosis

will be modified somewhat and arteriosclerosis without apparent pressure changes will be noted less frequently when the taking of diastolic pressure becomes more universal and less attention is devoted to the systolic pressure alone.

3. HYPERTENSION OF UNKNOWN ORIGIN. The third condition in which marked hypertension occurs comprises a group of cases in which all evidences of renal lesions are absent and which present no signs of sclerosis. The cause of the increased arterial tension in these cases is entirely a matter of conjecture. It is evident that for some unknown reason a spasm of the vessels occurs. Some have endeavored to show that this is due to increased adrenalin content of the blood, and a favorite hypothesis is that, if not adrenalin, then some other pressure-raising substance circulates in the blood. Even the recent exhaustive study of this condition by Riesman has failed to throw new light on its etiology. With the present uncertain state of our knowledge we had best be content with the dictum of Osler who states that it is more than likely that "the primary difficulty is somewhere in the capillary bed—in that short space in which the real business of life is transacted." The importance, however, of this form of hypertension lies in the fact that if it persists it is soon capable of producing hypertrophy of the heart, and later, through the constant wear and tear which it exerts on the bloodvessels, it leads to true arteriosclerosis.

It is of interest and perhaps of value to inquire how far the etiology above discussed coincides with actual clinical experiences, and further what clinical significance should be attached to extreme degrees of hypertension. To this end I have analyzed a series of fifty cases. A majority of these cases came under my observation through the kindness of the late Dr. John H. Musser, who afforded me ample opportunity to study them frequently; the remaining cases I have selected from my own and from hospital records. For the most part the patients were ambulant and represent types

frequently encountered in office practice. They were all under observation for periods varying from a few months to ten years. In nearly every instance repeated examinations of the blood pressure and urine were made. The earlier blood pressures were taken with the Stanton sphygmomanometer by the palpatory method, but the more recent ones were determined by auscultation with the Nicholson apparatus. Practically all blood pressure readings made during the past two years have been made by the auscultatory method. In order to keep off all debatable ground, I have selected only cases in which the average systolic blood pressure was 200 mm. Hg. or over.

First of all, I have grouped the cases according to their diagnoses which were made and recorded only after the patients had been carefully observed. As was to be expected, all fell under the three classes of cases to which I have just referred, *i. e.*, chronic nephritis, arteriosclerosis, and hypertension of unknown origin. In some a diagnosis of both chronic nephritis and arteriosclerosis was made, since both conditions were equally obtrusive and it was impossible to determine which was primary. This grouping showed that 27 cases had undoubted evidence of arteriosclerosis associated with more or less nephritis, 21 were primarily nephritics, and a much smaller number, only 2, could be classed as instances of simple high tension. In an analysis of 130 cases with a systolic pressure of 200 mm. or over, Janeway found that 105, or 81 per cent., were nephritics; 8, or 6 per cent., were doubtful cases of nephritis, and 17, or 13 per cent., were non-nephritic.

Taken by decades ranging from twenty to seventy years, my cases of hypertension were distributed as follows.:

Under 30 years	1
30 to 40 years	4
40 to 50 years	8
50 to 60 years	19
60 to 70 years	14
Over 70 years	4

It will be seen from this that the greatest number of cases occurred during the sixth decade. The few cases that occurred under forty years of age were all cases of nephritis, three of them being instances of chronic parenchymatous nephritis.

The distribution as to sex was: 31 males and 19 females. In the minds of many, hypertension is so largely associated with males that it may be surprising to discover how often the condition occurs in women. The women with this symptom were invariably past middle life, were almost always married, and a little over 50 per cent. of them had borne children.

It is traditional to lay stress upon the family history in cases of cardiorenal disease. In the histories of nearly all these patients this question had been asked. It was found that in almost one-half of the cases, in 22, either one or the other parent or some of the grandparents, had died of apoplexy or uræmia. In 1 case a brother had marked arteriosclerosis. It would seem from this that there is some justification for inquiring into the family history in our efforts to obtain a more complete knowledge of these cases of hypertension.

With the hope that the previous medical history might throw some light upon the etiologic factors underlying this condition of extreme hypertension, the records of these patients were examined with the view of determining whether or not they had suffered from any acute infections which might have sown the seed of subsequent cardiovascular or renal disease. In 3 instances no record of the previous medical history was given. Of the remaining 47, typhoid occurred 11 times; scarlet fever, 7 times; influenza, 5 times; rheumatic fever, 4 times; erysipelas, 4 times; recurring sore throat, 4 times; croupous pneumonia, twice; eclampsia, twice, and gout, twice. In 2 cases diabetes had antedated any evidence of nephritis, and peritonitis had apparently been a factor in 1 case. In 2 cases the previous history was negative.

Strange to say, definite luetic history was obtained only once. It seems to me probable that had a Wassermann reaction been taken on all cases of high arterial tension, lues would have been found to play a role in the etiology. However, in none of Riesman's cases of unexplained hypertension in women, was syphilis a factor. That the toxins of acute infections may cause degenerative changes in the arterial coats has been established and Thayer has shown that not infrequently typhoid fever leads to early arterial changes. It is, therefore, not surprising to find a number of cases giving a previous history of this disease. The relation of scarlet fever, influenza, erysipelas, acute articular rheumatism, and sore throats to nephritis, is too well known to require comment.

Alcohol, tobacco, overwork, worry, coffee, and excessive eating have all been blamed for the occurrence of arteriosclerosis. Of my patients, 13 had used alcohol to excess; an equal number were heavy users of tobacco; overwork and worry was apparently a cause in 17; while coffee and excessive eating were possible factors in only 2 and 8 cases respectively. Cabot doubts the importance of alcohol in arteriosclerosis, and Riesman could not attribute his cases to this form of toxæmia. Many observers are now prepared to regard tobacco as an important factor in producing arterial degeneration, and overwork and worry are well known as causative agents in the production of arterial spasm, hypertension, and ultimately sclerosis. From the above it will be seen that no one factor, whether infectious or toxic, can be accorded a dominant role in the production of high blood pressure.

It is well known that many individuals live in comfort for years with extreme degrees of hypertension without evincing any troublesome symptoms. When they do finally consult a physician, it is frequently for some symptom which off-hand would not be referred to the cardiovascular system.

A tabulation of the chief symptoms complained of by the patients in this series shows the following:

Indigestion	32
Dyspnoea	30
Headache	23
Palpitation	22
Weakness	17
Disturbed vision	15
Œdema	11
Angina pectoris	10
Vertigo and dizziness	8
Nervousness	6
Obesity	5
Insomnia	4
Tingling of extremities	3
Numbness of extremities	2
Asthma	2
Facial palsy	1
Purpura	1

The frequency with which gastro-intestinal symptoms bring the patient to the physician's office is well known. Riesman found that one-half to two-thirds of his cases of simple high tension complained of indigestion. He also found, as have I, that dyspnoea was a common symptom. It occurs apparently without any evidence of cardiac failure being present. Constipation was the rule and occurred in thirty-two of my series. Often it was extremely obstinate.

The occurrence of serious circulatory eye disturbances as a result of high arterial tension is well known. Indeed, in not a few of the cases here recorded, their general condition was first recognized by the ophthalmologist. Of my 50 cases, in 29 the ocular condition was normal, except for errors of refraction. In 10 albuminuric retinitis was present, retinal hemorrhages occurred in 4, and choked disk once; of these 15 cases, 9 died within three months to a couple of years after coming under observation. Subconjunctival hemorrhage occurred in 1 case, vitreous hemorrhage once, 1 patient

showed marked engorgement of the retinal veins only, in 1 glaucoma developed, and 2 had cataracts.

As was to be expected all but 4 cases showed marked cardiac hypertrophy with accentuation of the aortic second sound in most instances. In the majority of cases endocardial murmurs were absent, but various murmurs were noted in some. The diastolic murmur of aortic insufficiency was present 5 times. A systolic aortic murmur, unaccompanied by the signs of aortic stenosis, was of more common occurrence, and was observed 13 times. This murmur is commonly disregarded, but as Riesman points out, when it occurs in cases of this type in the absence of signs of a dilated aorta or aortic stenosis, it is an indication of sclerosis of the aortic arch and is of considerable prognostic importance, since it has been shown that the cases in which this murmur is present as the result of aortic thickening are the ones most prone to coronary sclerosis and angina pectoris. The murmur of mitral insufficiency is of rather frequent occurrence and was found in one-third of the patients. It usually indicated a relative insufficiency of the mitral valve secondary to hypertrophy and dilatation of the ventricle. In only 3 of my series was this systolic mitral murmur due apparently to true mitral insufficiency. A dilated aortic arch was present only 4 times, although x-ray examinations of the chest would probably have shown it to be a more common manifestation. Arrhythmia, usually of extra systolic type, was observed in 17 cases. Enlargement of the liver, not apparently due to circulatory failure, was observed 6 times.

The urinary analysis showed that in almost every instance albumin was present in variable amounts, although casts were absent more frequently. Even in the cases which from their histories were in all likelihood mostly arteriosclerotic, it was rare not to find some evidence of renal disease. In only 2 cases were albumin and casts both absent. This fact materially reduced the number of cases that it seemed justifiable to include in the group of causeless hypertension.

The specific gravity of the urine varied between 1002 and 1022, in only 4 instances did it exceed 1020, and it averaged 1012. Albumin varying in amount from large quantities in the several cases of chronic parenchymatous nephritis to the faintest traces, only detected by the potassium ferrocyanide test, was observed in all but 2 cases. Except in 1 case glycosuria was always absent. An excess of indican was observed only in 6 cases, in spite of the fact that constipation was rather the rule and that intestinal toxæmia is regarded by many as a common cause of arteriosclerosis. The truth of the matter is not that in these cases intestinal toxæmia did not exist, but that indicanuria is an unreliable index of this condition. Urobilin was also of rare occurrence and of doubtful significance. Casts were of less frequent occurrence than was albuminuria. Ten cases showed no casts on repeated examination of centrifuged specimens. In the remaining cases hyaline casts were uniformly present, and in 14 granular casts also occurred. The presence of the latter was associated either with circulatory failure or marked nephritis. Polyuria was noted in 23 cases.

These urinary analyses make it evident that in spite of much that has been said about hypertension unassociated with renal and vascular lesions, it is indeed a rare event to find this abnormality of the circulation present with constantly normal urinary findings.

Although blood examinations were routinely made in these cases, noteworthy anæmia was but rarely observed. Nine cases showed a hæmoglobin below 70 per cent. and only 4 had a hæmoglobin below 60 per cent.

Gastric analyses were made on 18 of the patients who complained especially of marked gastro-intestinal symptoms. Of this number 10 showed hyperacidity, but in 7 the condition found was one of hypoacidity and chronic gastritis, while in one case the gastric analysis was normal. The examinations of the stools revealed nothing of significance. Occult blood was found only twice, once in a case with

purpura, and in a second patient who suffered severely from hemorrhoids.

From the standpoint of both the patient and the clinician, one of the most important questions is the prognosis of these cases of grave hypertension. Of the total number of cases, 24, practically 50 per cent., are now dead. The shortest time that a fatal termination occurred after the patient was put under treatment was three months, the longest period during which a fatal case was observed was ten years. Uræmia was the cause of death in 7, apoplexy in 4, angina pectoris in 3, circulatory failure in 9, and pneumonia in 1. This analysis bears out the assumption that in these high pressure cases apoplexy occurs rather infrequently; it also confirms the fact that the most important factor in the prognosis of these cases is the condition of the heart. So long as the latter is capable of meeting the increased demands made upon it, the circulation is maintained with little functional disturbance. Sooner or later, however, degeneration of the hypertrophied cardiac muscle takes place, circulatory failure supervenes, and the well-known symptoms of decompensation appear. It is evident from the above that such extreme degrees of hypertension must always be regarded as of serious import, but with judicious and careful management the majority of these cases can be rendered at least temporarily more comfortable and certainly half of them may continue to enjoy a useful existence so long as their circulatory powers are conserved.

TREATMENT. The phase of this subject that is of greatest practical importance is the treatment. When high pressure first came to be recognized, there was a decided disposition to attempt to combat this symptom by active measures. Happily this tendency has yielded to a more rational conception of the process, and clinicians everywhere are learning to follow the teachings of Krehl and others, and are coming to regard high pressure as a conservative process. It has been

shown that in nephritis the hypertension increases elimination through the damaged kidneys and that in arteriosclerosis it increases the circulation in organs whose nutrition is impaired because of their diminishing blood supply. With this conception well in mind, it is evident that rational treatment should not be directed so much toward a mere lessening of the hypertension, but rather to removing if possible its underlying cause.

The treatment of hypertension falls under two heads: (1) Its prophylaxis, and (2) its management when once developed. In arteriosclerosis and nephritis, the chief causes of hypertension, it is more than ever true that "an ounce of prevention is worth a pound of cure."

How is prevention to be effected? By routine examinations of the blood pressure, taking both systolic and diastolic pressures in all patients in order to determine whenever possible the early signs of developing sclerosis. If we can detect the process when the elevation of pressure is only moderate and is due to vessel spasm, before anatomical changes in the vessel walls take place, therapy may be of avail. Having discovered in an individual approaching or past middle life early evidences of sclerosis with or without renal involvement much can be done to prevent the advance of the condition. Attention should be directed toward the patient's general hygiene and mode of life. Physical overexertion, great mental strain, and worries should be avoided. The patient should take a proper amount of recreation and rest; the latter particularly must be insisted upon. All sources of chronic infection as sinusitis, pyorrhea alveolaris, etc., should be removed; proper elimination should be insured by having the patient take several times a week hot air or electric light baths, or hot packs; and the bowels should be kept freely moved without causing actual purging. Of equal importance in these cases is the diet. Alcohol, coffee, and all irritating food and drink should be avoided and the quantity rather than the quality of the diet should be restricted. The intake

of nitrogenous foods should be reduced and the patient given an otherwise wholesome, sufficiently mixed diet. It will be noted, that at this stage drugs play no part in the treatment, except they be used to overcome intestinal stasis. If intestinal toxæmia is suspected or proved, high colonic irrigations two or three times a week are of value, and there are some who have claimed good results from the use of the Bulgarian lactic acid bacillus. As a result of the teachings of Lane, at present a popular intestinal antiseptic and laxative is found in paraffine oil, administered in cold water after each meal in doses of one-half ounce.

A second prophylactic factor in arteriosclerosis is the education of the patients themselves to the importance of easing up upon the activities of life, after they reach the age of fifty, the advantage of lessening the quantity of food taken as they grow older, and the avoidance of alcohol, etc., and finally the desirability of having their urine examined and blood pressure taken as a routine precautionary measure every six months.

The prevention of nephritis does not differ materially from that above outlined, since the causes of chronic nephritis are not dissimilar from those of arteriosclerosis with this exception: the frequency with which nephritis follows infections, even of minor grade, must not be lost sight of and it is incumbent upon the general practitioner to see that all cases with acute infections are kept in bed, their diet restricted, and their elimination adequately maintained; that every effort is made to prevent the occurrence of a nephritis; and that before the patient is finally discharged, it is definitely determined that the urine is normal and that no abnormal alteration has occurred in the blood pressure. Were these measures conscientiously carried out, there is no doubt but that many cases of chronic Bright's disease and arteriosclerosis might be averted.

The management of hypertension when its existence depends upon some well defined anatomical condition is

both difficult and unsatisfactory. Bearing in mind that the underlying cause is usually toxic and that the hypertension is compensatory, our chief effort must again be toward elimination and lessened production of all toxic and irritating material. The most useful measures are comprised under the following:

Rest. No more efficient method exists for lowering blood pressure than prolonged rest in bed. For example, one of the patients in this series, whose systolic blood pressure had averaged 230, improved markedly subjectively and had a systolic pressure of only 170 after two weeks' rest in bed and while on a bland diet.

Dietetic. As in the prophylactic treatment, the diet should be limited in quantity particularly, especially since over-eating has frequently been a fault in many of these cases. The nitrogenous intake should be restricted, and all stimulants, especially alcohol, coffee, and tobacco, should be avoided. The patients should be instructed to eat regularly, slowly, to chew well, to rest one-half hour after meals, and to take their heartiest meal at midday.

Elimination. Aside from the kidneys the chief avenues through which additional elimination may be obtained are the bowels and the skin. Violent cathartics are contraindicated, but several free bowel movements should be obtained daily by the use of such laxatives as senna, rhubarb, cascara, or milk of magnesia. The addition to the diet of stewed fruit is of great help in this respect. In some cases the salines in moderate doses seem most efficient. A good practice is in addition to have the patient take a dose of blue mass (gr. 5 to 10) followed by a saline every two or three weeks.

Various forms of sweating have been devised in order to increase the activity of the skin. Excessive sweating is frequently harmful to many of these cases and the steam room of the Turkish bath is decidedly depressing and dangerous. Short electric light baths carefully given, or hot packs, or even simple hot baths are usually sufficiently vigorous meas-

ures. In beginning treatment it is well to have the patient take such a bath every day for a week, then the next week every other day, and finally two or three times a week as a routine procedure. The value of these baths is not that they cause any permanent or marked lowering of the pressure (for it has been shown that the lowering is only moderate while the baths are being taken and that the blood pressure soon returns), but that they cause increased elimination by inducing cutaneous hyperemia which in turn lessens the congestion of the splanchnic and other great visceral vascular areas.

Certain newer forms of therapy, such as the high frequency current, are enthusiastically spoken of by some. Personally I have found but meagre evidence for placing faith in the value of such measures.

A procedure of undoubted value in the management of certain cases of high tension is venous section. This method does seem to cause at least a temporary lowering of pressure and may be practised with safety, provided the amount of blood withdrawn is controlled by frequent blood pressure readings. Twelve to twenty ounces can usually be taken with impunity. After a venous section in my own experience, and the same was found by Miller, blood pressure is only lowered for a short time and in cases of uræmia which are bled the pressure is often lowered little if at all. The good that results in these cases may be due to the withdrawal of a certain amount of toxic material.

Drugs. Finally we may turn to a consideration of drugs in the treatment of hypertension. The widespread and promiscuous use of the nitrites in high blood pressure must be regarded as a misdirected therapeutic procedure. Too many of us have fallen into the habit of giving some form of nitrites whenever hypertension is encountered. Much careful work has of late been done on this subject and it seems well established that not only do the nitrites fail to do any lasting good, but in a large number of cases, if we accept the com-

pensatory conception of hypertension, they are capable of positive harm.

That nitrites lower blood pressure by dilating the vessels is undoubted, but that this lowering is decidedly fugacious is equally certain. It has also been shown that when nitrites are used over any length of time, in ordinary doses their vasodilator effect becomes *nil*. In a series of careful observations Miller showed that when either nitroglycerine, erythrol tetranitrate, or sodium nitrite were given patients, a fall of blood pressure strikingly similar in each case resulted in two to four minutes, but that in each case this fall was but temporary and that in two hours the blood pressure had regained its previous level. From this it is evident that these vasodilators find their chief use when it is necessary to combat some sudden condition of spasm such as angina pectoris or nocturnal dyspnea, but that they are inefficient as routine means of treatment.

In my series nitroglycerin was employed at some time in 27 cases. Of this number it seems to have had a beneficial effect on the symptoms in but 3 cases, while in 5 others its advantage or disadvantage could not be determined, and in the remaining 19 cases it had apparently no influence. In 1 case in particular, taking glonoin, a persistent lowering of the systolic pressure to 40 mm. and the diastolic pressure to 30 mm. occurred. Whether this was due entirely to the drug is difficult to say, since other therapeutic measures were being employed at the same time.

In contradistinction to vasodilators, and paradoxical as it may seem, distinct benefit seems to have resulted from the use of digitalis in some cases. Cushny has amply demonstrated that, except in conditions of low tension and circulatory failure, digitalis does not raise blood pressure. This drug may, therefore, be employed in small doses with safety in these cases. It does good chiefly in those patients in whom cardiac weakness is developing and often is wonderfully efficient in relieving the dyspnœa and vertigo. In my series

8 cases were markedly helped symptomatically by the use of digitalis, but in 4 instances these patients were showing some indication of decompensation.

Aconitine, in the dose of gr. $\frac{1}{240}$, 4 times a day, relieved a persistent headache in one patient with a blood pressure of 220. On the other hand, a patient whose blood pressure has for years been over 230 can take for weeks 90 minims a day of the tincture of aconite without any noteworthy drop in the pressure occurring, although the throbbing and pain in her head are apparently somewhat benefitted.

The iodides have always enjoyed a reputation in the treatment of hypertension. Their exact mode of action is not clear and their use is largely empirical. Perhaps the secret of their usefulness will be revealed when Wassermann reactions are done more frequently in these cases of high arterial tension. In view of the fact that antisiphilitic treatment does seem to help some cases in which lues is the etiologic factor, the Wassermann reaction assumes an importance from the standpoint of both prognosis and treatment.

All things considered, it must be admitted that in well-developed hypertension, be it of vascular or renal origin, efforts to lower it are of but little avail. In the 50 cases here analyzed, in only about 14 can it be said that the various therapeutic measures employed had much effect on the blood pressure. On the other hand, there is little doubt but that the general hygienic treatment of these patients, relieving them from physical exertion and mental anxiety, regulating their digestions, and promoting their elimination, was a most important factor in adding to their comfort and and prolonging their lives, even though the hypertension itself was unaffected.

SOME CASES OF NEURITIS AND NEURALGIA OF VARIED TYPE AND CAUSATION¹

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It is not the intention in this paper to discuss the pathology of the conditions, but to describe some cases which are of interest and which have been instructive.

The first was a Polish patient, aged about forty years, who had worked for only six weeks in a lead works, as a laborer. He was admitted to the Episcopal Hospital under the following condition: He was decidedly anæmic, the typical blue line was present on the gums. The bowels were moderately constipated, and he had a moderate degree of lead colic. The affection of the nervous system was most marked and the most interesting feature of the case. He had complete loss of power in the muscles of both shoulder girdles, and the triceps and extensor muscles of the forearms were entirely powerless. He had a complete preservation of power in the flexors of both arms. Had it not been for the history of his occupation, and the blue line, the case would on first glance have appeared to be a typical one of muscular dystrophy. As soon as it was possible he was transferred to the Orthopædic Hospital, where the reaction of degeneration was found to be present in all the affected muscles. He was given a long course of treatment, including potassium iodide, galvanism and massage. Owing to his complete inability to contract

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the muscles of the shoulder girdle he developed ankylosis of both shoulder joints, and it was necessary to break up the adhesions under a general anæsthetic. This is an unusual complication, and one that should have been provided against by regular passive movement of the arms. He left the hospital in the following condition: He had normal power in all the muscles, except the extensors of the forearms, which were slightly weak. The reaction of degeneration had been replaced by a normal reaction in all of these muscles. The lead line of course persisted, as is always the case.

The next patient was one of herpes zoster, occurring in a woman, seventy-three years of age. Her husband had died in May, 1910, and she had the readjustment of her life to arrange, in addition to her very engrossing direction of several large charitable organizations. In October, 1910, these combined strains upon her nervous system caused, as is often the case, the occurrence of this most painful malady. The seventh left dorsal nerve was affected.

The modern conception is that herpes zoster is due to an infection corresponding to acute anterior poliomyelitis, affecting, however, the posterior roots and nerves, instead of the anterior. The general condition did not seem to be markedly affected in this case, except by the pain, and the loss of sleep occasioned by pain. There was no fever, nor other evidences of a general infection. Various sedatives were used, with little avail, such as aspirin, phenacetine, gelsemine, bromides, and chloral, and finally it was necessary to resort to the use of codeine and morphine.

About November 30, the patient gradually developed a mental condition, which was described by Korsakoff, the characteristics of which are as follows: The patient is conscious, but is completely disorientated, both as to time and place, with very striking disturbance of memory. The memory of events and persons of the former life replace present events and persons with whom the patient is associated. About March, 1911, the patient's mentality became better,

and a complete restoration of all her mental faculties resulted. It has been impossible for me to account for the psychosis which developed in this case. It occurred too long after the onset of the herpes zoster to have been due to an infection, if an infection caused the neuritis in this case. Her elimination was very carefully watched and there was no evidence of intestinal auto-toxæmia or renal deficiency, which could explain its development. It may have been produced by the large amount of sedatives which were required, not only to provide sufficient sleep, but to reduce the intolerable pain which was wearing out her strength. The pain by June 1, although still present, was very much reduced, and still further subsided during the summer. It is now at this time, seventeen months following the onset, still occasionally present, particularly when the patient is overtired, or on turning over at night. It is very much milder and causes very little discomfort.

The next case illustrates one of multiple alcoholic neuritis. Alcohol is one of the most common causes of neuritis, as is well known. This case differs from the ordinary case, in that there was very little loss of muscular power. The patient was thirty-eight years of age, a resident of South Carolina, who had his first attack of neuritis in June 1910. He had been drinking heavily for some months, as a result of the pressure of his work. He began in May to have numbness of the feet, a loss of appetite, and a general physical depression, resulting from the large quantities of alcohol that he drank. He was admitted to the Orthopædic Hospital, June 20, 1910, and at this time had analgesia, or loss of pain sense, but was not anæsthetic nor hyperæsthetic. The feet and legs up to the knees were the only parts affected. There was slight paralysis of the anterior tibial muscles in this attack, but this was shown simply in a very slight weakness in the extension of the feet. There was no toe-drop. The knee jerks were preserved, but much reduced. Another abnormality of this patient was that there was no pain present in this attack, and only very slight tenderness over the nerve

trunks. Under rest, free sweating, iodides, and strychnine, together with massage and electricity, he recovered and was discharged cured in July, 1910. He had a second attack in April, 1911, following another hard spell of drinking, and was again admitted to the Orthopædic Hospital. This time his attack was longer, as it required about six weeks to correct the condition. The conditions in this attack were similar, except that the analgesia of the feet was much more marked, and paræsthesia and slight pain were present. There was, however, no muscular weakness. In August, 1911, he began drinking again. A fire destroyed several buildings in which there were lead tanks used in the business, and he was obliged to handle lead. He suspected that lead was an element in the causation of the third attack, which developed in November. This attack was also different from the ordinary one in the suddenness of the onset. On November 15, after drinking a glass of ice-water, he had a sudden feeling as if a heavy ball had dropped into his stomach, which persisted until about the first of January. In the same day, November 15, he noticed diminished sensation over the epigastrium, and later this appeared over the sternum. Still later sensation lessened in the legs. December 1, he had slight pain in the thighs. There was no blue line present, the bowels were regular, he was not anæmic, and there was no paralysis. Therefore, the possibility of lead being a contributing cause was eliminated. In this attack there was no loss of power, and the reflexes were lessened, but not absent. There was very marked analgesia of the dorsum of the feet, the legs, the thighs, and also in the epigastric region. There was no hyperæsthesia, and but very little pain. Under the same course of treatment the conditions improved so that he was able to leave the hospital on January 15, after six weeks of treatment. In spite of the fact that this man had been using large quantities of alcohol, he stated that it had never intoxicated him, and the withdrawal did not develop any symptoms of delirium tremens, although he stated that he had consumed

a quart of whisky between Washington and Philadelphia. There was no appreciable let-down when he was given but three ounces of whisky in the twenty-four hours, and he slept soundly all night on a small amount of sedative. It is well known that alcoholic neuritis causes primarily an affection of the sensory nerves, but there is almost invariably some motor involvement, most commonly of the anterior tibial muscles, which causes the steppage gait, and of the extensor muscles of the forearms, causing wrist drop. This is particularly likely to be the case in repeated attacks. There is usually marked pain, paræsthesia, hyperæsthesia, and tenderness over the nerve trunks, in addition to the analgesia, which was present in this case.

Another type of neuritis, which is not uncommonly seen, is due indirectly to alcohol. This is the type which occurs in intoxicated individuals who lie down in a drunken stupor, using their arm as a pillow to support the head. The pressure on the musculo-spiral nerve, which results, causes wrist drop, due to paralysis of the extensor muscles of the forearm, which usually appears promptly the next day. This clears up rapidly under counter irritation and faradism.

The type of neuritis resulting from more continued pressure is slower to cure, as in illustrated by this case. A waiter, aged about fifty-five, had fallen and sustained a Pott's fracture of his right leg. The ankle joint was quite stiff following the removal of the cast, and he was obliged to use a crutch for three weeks. At the end of this time he presented himself with a typical crutch palsy of the right musculo-spiral nerve. This cleared up, but only after six weeks of galvanism and massage.

Still another type of multiple neuritis, and one not very commonly seen, is that following diphtheria. The multiple neuritis which occurs after this infection differs from the alcoholic in that it does not usually cause anæsthesia or analgesia, but causes a loss of muscle sense similar to that observed in locomotor ataxia. Also, as is well known, the

bulbar nerves are likely to be affected, causing difficulty in swallowing, with regurgitation of fluid through the nose and attacks of choking. The following case is quite an interesting one in view of the onset. The patient, a girl of six, was brought to the Orthopædic Hospital on March 9, 1906, with the following history: She had had an attack of diphtheria on February 5, 1906, and antitoxin was administered February 5, and again on February 6 or 7. It was said the attack was severe, and the throat sore for one week. She went out first on March 2, and again on March 4, for a short walk. On March 5, she complained of pain in the soles of her feet. She was taken this day to see the family physician, and then to a department store. On the way home the child complained of being very tired and stumbled. She choked on attempting to swallow. The next day she was most ataxic and had difficulty in swallowing. There was no change in sensation in this case, the station was bad, and the gait was ataxic, being of a cerebellar type. The neck muscles were weak, and the head was held up with difficulty, but there was no paralysis of any muscles. The speech was of a bulbar type, and she drank with difficulty, but had no regurgitation. The pharyngeal reflex was present, but the movements of the muscles were weak. The knee jerks were absent. Dr. Holloway reported that there was no paralysis of the extra- or intra-ocular muscles. This case did not return to the hospital, so that nothing is known concerning the progress of the case. Usually under the use of electricity and increasing doses of strychnia a complete recovery results.

Local neuritis affecting but one nerve is a more common condition than is multiple neuritis, but the following are cases the causes of which are slightly unusual:

The first was a railroad freight conductor, who was treated at the Orthopædic Hospital, with the following history: In September, 1910, on a very hot day, a thunderstorm suddenly occurred, and he put on over his shirt a thin rubber coat. Being exposed to the full force of the shower, he became

quite chilled. Next day he began to have pain in his left shoulder and weakness of the left arm developed in a week. He had a neuritis affecting the left musculo-spiral nerve. The muscles affected were the triceps, extensor communis digitorum, extensor carpi ulnaris, lumbricales, and abductor minimi digiti. In spite of prolonged massage and galvanism there is still at present decided weakness in the extensor communis digitorum, the extensor carpi ulnaris and lumbricales, and the reaction of degeneration persists in these muscles.

Neuritis due to syphilis is not uncommon, but the nerve usually affected is the ulnar. This is particularly seen in the early stages of locomotor ataxia, and the occurrence of ulnar neuritis should always direct attention to the possibility of the development of this disease. The patient was a steam-fitter, thirty-five years of age, who admitted having had several attacks of gonorrhœa, but denied syphilis. There were never any secondary symptoms. In July, 1911, he had an attack, characterized by swelling of both ankles, and pain in both legs and feet. This lasted three weeks and when he began to walk he had considerable pain, and then noticed that his left leg was weak. He presented himself at the Orthopædic Hospital on October 26, 1911, in the following condition: His station was good, his knee jerks were present and active, and his pupillary reflexes normal. No Babinski was present, and the achilles jerk was normal. There was present a typical step-page gait affecting the left leg only. This leg was lifted high to clear the toes from the ground. Power in the left thigh was good, but there was complete loss of power in the peroneals, the anterior tibial, the extensor communis digitorum, and the extensor hallucis of the left leg, the muscles supplied by the external popliteal nerve. There was no anæsthesia or analgesia affecting the left leg. The left calf measured one inch less than the right. One of the physicians who saw him expressed the opinion that it was a beginning muscular dystrophy, which often occurs at this age. The

Wassermann reaction was positive, on November 1, 1911, and mercurial treatment was instituted, and he was given massage and faradism; the electrical reaction being normal. On November 18, he was given three decigrams of salvarsan. There had been very little improvement prior to this time. Immediately following the injection there was a decided improvement in the condition of the leg, and it was possible for him on the second day following to contract slightly the anterior tibial and extensor muscles of the toes, but the peroneal muscles were powerless. Mercurial treatment and electricity were continued, and on December 9, he was given a second injection of three decigrams of salvarsan. There was quite a sharp reaction following this, characterized by fever and general malaise, with some nausea. Two days following this there was distinct improvement evident in the ability to contract slightly the peroneal muscles, and more power was present in the other affected muscles. When last seen, February 8, he reported that he had been working three weeks. That he had had only three electrical treatments in the past month, but that he had continued taking his mercurial treatment. At this time there was a difference of about three-eighths of an inch between the two calves. His gait and station were entirely normal, and power had fully returned in the affected muscles. It was particularly instructive to see the effect of the much-discussed remedy, salvarsan, in this case. The improvement following each injection was so prompt as to convince me that it was most efficacious in this case. I am of the opinion, however, that this man should continue his mercurial treatment for a year or eighteen months, as he had not been treated previously.

The following case of supra-orbital neuritis is one which has been of much interest to me. The patient, a man of seventy-one years, developed in March, 1911, a very severe attack of supra-orbital neuritis. He had violent pain over the right eye, and the area of the distribution of the right supra-orbital nerve was so extremely sensitive that he was

unable to wear a hat or to tolerate the slightest touch, and was unable to comb his hair. Jarring the body by violently closing a book would cause a paroxysm of pain. There was at this time a copious purulent nasal discharge. The physician attending him was a very able and experienced clinician, but he did not discover the cause until the patient was seen by an oculist. The latter suggested calling a rhinologist to investigate the condition of the accessory sinuses. This disclosed an infection of the right frontal sinus. Under appropriate local treatment he recovered. There was a second attack of this condition in September, 1911, due to cold and raw weather, a third in November, a fourth in December, and a fifth developed on February 11. The last followed an exposure for a considerable time to extreme cold and high winds the previous day. A treatment of the nose on February 14, decidedly relieved the condition, although the nerve was still tender, and there were continued paroxysms of pain. On February 19, the condition was much relieved. In the attacks in November, December, and February, while under my observation, there was no nasal discharge.

Cases of neuralgia due to anæmia, neurasthenia, nephritis, chronic rheumatic states, and eye strain, are relatively common. A few cases of neuralgia due to some rather unusual causes are here presented:

The first, a man of fifty years, began in November, 1909, to have pain back of his right ear, in the right side of his neck and in the right shoulder. This became very severe after December 15, 1909, and when seen November 11, 1910, he stated he had never been entirely free from it. He had been treated by other physicians with various remedies, but with very little relief. When the area in which the pain was present was examined, with parts uncovered, a decided expansile pulsation was observed in the right supraclavicular fossa. A loud bruit was heard over this area. There was an area of dulness above the second rib, extending from the right border of the sternum to the midclavicular line. The

heart was enlarged, and there was a rough systolic murmur at the apex and at the base. There was equal pulsation in the radial and temporal arteries, and there was no tracheal tug. The blood pressure was 200. There was a history of rheumatic iritis twenty years previously, and the rheumatic infection was assumed to be the cause of the existing arterial disease, which had resulted in an aneurism, supposedly of the right common carotid artery. Dr. John B. Deaver operated in December, 1910, and found an anomalous *left* common carotid, which wound back of the trachea, and which was the seat of a fusiform aneurism. He was unable to ligate the artery, and was obliged to limit the operation to an exploration. For one week after the operation the pain was very much relieved, but it soon returned and became very severe.

The following case of tic douloureux was not different from other cases of this most serious disease, but it is interesting from the standpoint of treatment. The patient was a man of forty-two years, when he was first seen by Dr. Wharton Sinkler, in 1906. He had always been gouty, and while not using stimulants to excess, had indulged rather freely. In 1905, he began to have neuralgia in the right side of his face. This first affected the infra-orbital branch of the fifth nerve. Later it extended into the jaw, the roots of the teeth, and the right side of the tongue. The amount of his work and weather conditions made no difference. Talking, eating, brushing the teeth, or any movement of the muscles produced an attack of violent pain. The pain was intense and there was present twitching of the face during the paroxysm. There was tenderness over the supra- or infra-orbital nerves, or over the mental foramen. Under large doses of strychnia and anti-spasmodics, he kept fairly comfortable, not having many or severe attacks until December, 1908. In April, 1909, he was injected by Dr. Kiliani, of New York, and was comfortable afterward until January, 1910. He began about January, 1910, to have gradually increasing

attacks of pain, which were not relieved by any form of treatment. On my advice he went to Chicago and was injected by Dr. H. T. Patrick on February 28, 1910. It required three injections before Dr. Patrick struck the nerve, and there was immediate analgesia and paræsthesia of the cheek, tongue, and lips. The seventh nerve was paralyzed as a result of this injection, this being due to a parotitis of the right parotid caused by the injection. The facial paralysis, due to the injury of the seventh nerve, lasted until June, 1910. When examined last in October, 1910, there was still paræsthesia of the lower lip and chin, extending along the jaw and up to the ear. Up to January, 1912, there had been no attack of tic douloureux since the injection in February, 1910. These cases are so serious that many of the sufferers have been obliged to take opium for relief, and have acquired that most demoralizing habit. The injection treatment as practised by Dr. Patrick affords relief for a period, varying from a few months to five years. There is nothing in the procedure, or in its results, which prevents a second injection being performed and affording another period of relief. The injection is a much less serious matter than is the radical operation of the removal of the Gasserian ganglion, and the latter in many cases affords relief for no longer a time than does the injection.

While neuralgia due to malaria is not uncommon in certain sections of the country, it is by no means a common condition in Philadelphia. The following case was that of a machinist, aged twenty-three, who was seen on July 26, 1909. He lived in Philadelphia, and had been born and spent all his life here. He stated that each year for seven or eight years there had begun in June and lasted until August, a pain over his left eye and left side of his face. The pain was constant, becoming gradually more severe, and he had been obliged to stop his work for several weeks. The pain had begun on July 1, 1909, or nearly four weeks before he was seen, and was so severe as to prevent his working. He stated that when the pain was most severe there was a discharge from

the left nostril, but no catarrh at other times. There was tenderness over the left side of the face, but no marked tenderness over the frontal sinus. On close questioning he stated that he had had malaria when he was seven or eight years old. He was given a pill of Warburg's tincture, and on August 1, six days later, he wrote that the neuralgia had been cured. In the absence of definite chills, and without finding the plasmodium malariae, it is not very scientific to make a diagnosis of malaria. But in this case the fact, that although the neuralgia had been present for four weeks, it was promptly cured by antimalarial treatment would make it seem reasonable to assume that malaria was the cause. In addition neuralgia of such duration due to other causes does not clear up so promptly on the use of salicylates or quinine.

The two following patients were almost exactly similar. The first, a woman of sixty-five, following an attack of tonsillitis, bronchitis, and considerable acute rhinitis, on March 28, 1911, developed one month later on April 25, 1911, much pain, principally in the right temple and across the forehead with a feeling of compression in that region. There was no nasal discharge, and it was at first believed that the neuralgia was due to arteriosclerosis, since the blood pressure was 220 and has continued at about that figure. After trying various forms of treatment without any affect, she was referred to Dr. F. R. Packard, who found an acute ethmoiditis, and a marked congestion and swelling of the turbinates, which were pressing against the septum. He found in addition deep crypts in the tonsils, which contained plugs similar to those seen in follicular tonsillitis. His local treatment finally relieved this attack, but there have been numerous attacks during autumn and winter, since the onset of cold weather, which have been relieved by treatment of the nose and by the removal of the plugs from the tonsils.

The second case was a woman of forty-three, and was referred on December 20, 1911, by Dr. John H. Bromley. This was a case of severe neuralgia, of the left side of

the face, which occasionally extended slightly to the right. She had not been benefitted by a careful refraction. There had been no acute rhinitis, and the patient declared there had been at no time any discharge from the nose. There being no diseased teeth or other local conditions which would explain this pain, she was referred to Dr. George B. Wood, who reported that on passing a probe between the middle turbinal and the septum toward the sphenoidal sinus of the left side, several drops of pus oozed out along the probe, and he was subsequently able to wash out a large quantity of pus from the left sphenoidal sinus. The pain was immediately relieved, and when seen three days later, she declared that there had been no pain since Dr. Wood had treated the nose. The tenderness had entirely left.

There are similar cases of neuralgia, but due to an infection of the antrum of Highmore. A number of these cases have been observed by the writer and in each there was an acute coryza with muco-purulent discharge. Violent pain and tenderness throughout the affected side of the face was present. In most of them the passage from the nose into the antrum was obstructed, and the pain was relieved only by continued local treatment to secure drainage of the antrum. A short sojourn at the seashore or in the pine woods of New Jersey has effected a prompt cure in most cases, after the need for local treatment had passed.

REPORT OF A CASE OF HERPES ZOSTER COMPLICATED BY BELL'S PALSY¹

BY HORACE E. HAPPEL, M.D.
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It seemed best before taking up the history of this case, briefly to mention a few points in the anatomy and symptomatology of the conditions to be considered.

The seventh nerve has its origin in the floor of the fourth ventricle near the nucleus of the abducens. Before it leaves the preoblongata it is joined by fibres from the nucleus of the third nerve, which are now considered to supply the anterior belly of the occipitofrontalis, the orbicularis palpebrarum, and the corrugator supercilii muscles.

The seventh nerve passes out upon the mid-peduncles of the cerebellum and into the internal auditory meatus with the auditory nerve, from which it receives a branch, the *pars intermedia*. Then it passes through the aqueductus Fallopii in the petrous portion of the temporal bone and emerges from the stylomastoid foramen. It passes forward into the substance of the parotid gland, crosses the external carotid artery and behind the angle of the mandible divides into branches, forming the *pes anserinus*, which supply the muscles of expression.

The chorda tympani is given off 5 mm. from the point of exit at the stylomastoid foramen. This nerve is concerned in the act of hearing through its branch to the stapedius

¹ Read at a meeting of the Episcopal Hospital Clinical Society, March 18, 1912.

muscle, and in the sense of taste in the anterior two-thirds of the tongue, and in the flow of saliva.

As the facial nerve emerges it gives off branches to the stylohyoid and posterior belly of the digastric muscles. Communicating branches join the great auricular, small occipital and superficial cervical nerves, branches of the superficial cervical plexus.

A destructive lesion of the facial nerve causes paralysis of the muscles of expression. *If the lesion is central*, the anterior portion of the occipitofrontalis, orbicularis palpebrarum and corrugator supercilii muscles escape paralysis, because they are supplied by nerve fibers which have their origin in the nucleus of the motor oculi. There is usually an associated paralysis of the abducens, with hemiplegia of the opposite side. There is no reaction of degeneration in the paralyzed muscles, since the lesion does not lie in the course of the nerve.

A lesion in the petrous portion of the temporal bone would cause loss of sense of taste in the anterior two-thirds of the tongue, dryness of the mouth, and impairment of hearing, because of involvement of the chorda typani.

After exit from the stylomastoid foramen, all the muscles of expression are paralyzed except the levator palpebræ (supplied by the third nerve), the posterior belly of the digastric and stylohyoid, to which branches are given off just before it emerges.

SYMPTOMS OF PALSY. Smoothness of forehead, due to palsy of occipitofrontalis. Inability to frown, from paralysis of corrugator supercilii. Eyes cannot be closed, on account of involvement of orbicularis palpebrarum. Obliteration of nasolabial groove, from paralysis of levator anguli oris.

Smoothness of cheek and drawing of mouth toward sound side, from inability of the muscles of the affected side to resist those of the opposite side.

Patient is unable to dilate the nostril because of palsy of levator labii superioris alæque nasi.

He cannot whistle on account of loss of function of the orbicularis oris, and food collects between the gums and cheek because of weakness of the buccinator.

As to the nerve supply of the parts affected by the herpes zoster in this case, we find that the anterior portions of the second and third cervical nerves, through their branches, the small occipital, great auricular and superficial cervical, supply the skin of the anterolateral portion of the neck, that of the parotid region of the ear, and postauricular region.

The posterior portions of the second and third cervical nerves, through their branches, the great and the third occipital, supply the integument of the occipital region extending upward to the vertex.

J. P., aged fifty-eight years, native of England, a weaver by occupation, admitted January 11, 1912, to the Episcopal Hospital, in the service of Dr. Frazier, with provisional diagnosis of carbuncle of the neck and scalp.

Family History. Negative.

Personal History. Negative, except that for the past two years the patient has had vague pains in limbs, which he ascribed to rheumatism.

Twenty-eight years ago he was struck on the left side of the face, sustaining a fracture of the nose and an injury to the eye, resulting in impairment of vision. Patient denies syphilitic infection, but had gonorrhea in young manhood.

Present Illness. Three weeks prior to admission, December 21, 1911, the patient contracted a cold in the head, had severe pain on the left side of the neck and scalp, and on the following morning discovered a vesicular eruption on the left side of his neck, cheek, and occipital portion of the scalp. The contents of the vesicles became purulent, the vesicles ruptured, and thick crusts were formed. His family physician was consulted and he prescribed an ointment, which was without effect. The suppurative process increased in extent, but at no time crossed the median line.

The patient suffered considerable pain, had insomnia, anorexia, and constipation. About one week prior to admission, he noticed that the left side of his face was paralyzed. His condition showing no improvement he was advised to go to the hospital and was brought in the ambulance January 11, 1912.

Condition on Admission. Slightly emaciated, pallid, anæmic-looking man, aged fifty-eight years.

Head. Nose displaced to right side of face; left eyelid red and slightly swollen; pupil irregular and contracted, probably due to an old iritis; conjunctiva congested; vision limited to object perception; right eye shows incipient cataract; complete facial paralysis of left side of face; impairment of hearing in left ear, ascribed by him to a cold in the head.

From the median line anteriorly, the left side of the neck, the parotid region of the cheek, the occipital region of the scalp up as far as the vertex and extending to the median line posteriorly, were covered with masses of yellowish-brown crusts, beneath which pus could be seen. The tissues were indurated and tender. A few reddened papules were present on the root of the neck. Tongue dry, fissured, and coated with a brownish material.

Chest. Negative.

Abdomen and Extremities. Negative.

The condition was regarded as herpes zoster, with secondary infection. Smears of pus showed pus cells, diplococci, and micrococci. *Staphylococcus pyogenes aureus* obtained in pure culture from pustules. Wassermann reaction was negative.

Treatment. Patient was put to bed, left side of scalp and beard shaved, crusts removed with olive oil, parts cleansed with peroxide, soap and water, and unruptured pustules were punctured and interior swabbed out with pure carbolic acid, followed by alcohol. Dressings of ammoniated mercury ointment spread on lint were applied.

Laxative and tonic ordered.

January 15, 1912. Patient looks and feels better. Dressings removed and parts cleansed. Appearance improved. No new pustules. Many have healed and others are disappearing. Ammoniated mercury ointment re-applied.

January 17. Parts cleaner. No new pustules. Considerable induration remains.

January 19. Induration lessening. Pus evacuated from two collections and carbolic and alcohol employed. General condition improved.

January 23. Parts look much better. Induration disappearing.

January 26. Neck well, save for a few indurated areas.

February 1. Skin smooth, no induration, general condition good. Patient complains of pain beginning at angle of jaw and extending over left side of face. Electricity ordered.

February 29. No improvement noted in palsy. Patient still has pain, but probably not so severe.

CONCLUSIONS. The herpes zoster in this case probably was due to an acute inflammatory lesion, involving the ganglia of the second and third cervical nerves.

The connection between the herpes zoster and Bell's palsy is not clear. It may have been due to the same cause as the herpes, or the ulceration at the angle of the jaw may have involved the facial nerve, or it may have been a coincidence.

The fact that there are communicating branches between the second and third cervical nerves is a matter of interest, but could hardly be offered as a plausible explanation of the facts in this case.

REPORT OF A CASE OF MYOPATHY¹

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THE case which I wish to report tonight is one of myopathy. This disease is divided into three principal types: The pseudomuscular hypertrophy; the scapulohumeral, and the facio-scapulo-humeral types.

In the pseudomuscular hypertrophy the disease begins in the muscles of the calves and back, usually between two and twelve years of age; and many of the muscles show a marked increase in size at first.

In the scapulohumeral type, the disease begins in the muscles of the shoulder-girdle and upper arm, and the muscles decrease in size from the beginning. It starts usually from fifteen to twenty years of age.

In the facio-scapulo-humeral type there is an early involvement of the face muscles in addition to those of the shoulder-girdle and upper arm. This type usually begins earlier than the scapulohumeral.

There are other minor types which, however, are not as distinct as these. But even those types whose features are most pronounced do not keep entirely distinct, and mixed forms are frequently met with.

W. P., aged seventeen years, single, born in the United States, parents also born in United States. On the mother's side: Grandmother, aunt and uncle died from phthisis; grandfather

¹ Read at a meeting of the Episcopal Hospital Clinical Society, March 18, 1912.

died from intussusception. On the father's side: Grandmother paralyzed several years before death; grandfather died from a stroke before fifty years of age; uncle has locomotor ataxia with optic atrophy. Father living and very nervous and delicate. Mother living and well, of frail appearance; married at seventeen years, no miscarriages. Brother, aged twenty-four years, has pseudomuscular hypertrophy, which began at twelve years; brother, aged twenty-two years, healthy; brother, aged nineteen years, has pseudomuscular hypertrophy, which began at eleven years; brother, aged twelve years, has pseudomuscular hypertrophy, which began at five years. No other cases in earlier generations.

Normal birth, no instruments, breast fed. Healthy as a baby, but very nervous. Slow in cutting teeth. Began to walk at the age of thirteen months. Talked at the age of two years. Pertussis at three years. Measles at five years. Typhoid fever at eight years. Mother states that he has always been very anæmic. During the past five years has had several attacks of "rheumatism;" never had to go to bed, pains in arms and legs, especially the jaws. Began work at the age of fourteen years in a stocking mill, as a turner. For the past two and one-half years has been working in a carpet and tapestry mill. Work consists of changing beams and warps weighing from 50 to 150 pounds, with the help of another boy.

Seen first about five years ago and treated for anæmia and joint pains, apparently rheumatic; mitral systolic murmur at this time. No atrophy noted, although patient was examined stripped, but a beginning atrophy may easily have been overlooked, as I did not especially look for it.

The family are very sensitive about this trouble and did not tell me anything about it until later, when I treated one of the other boys for tonsillitis.

Next seen a few weeks ago, when the patient had been sick for a few days with coryza, general malaise and fever. It was at this time that I noticed the atrophic muscular condition.

Examination. Well-grown boy. Anæmic. Lips thick, are kept separated and more or less everted; can be closed, however, but can whistle only with difficulty. States that he used to be able to whistle easily but has not done so for some time. Labionasal fold very faint. Other muscles of the face appear normal.

On being stripped, arms, chest, and shoulders look emaciated. The shoulder blades are scaphoid and the vertebral border pro-

jects prominently, tending with the protruding belly to exaggerate the apparent deformity of the lordosis which is present.

The muscles of the shoulder girdle, upper arms, and back, except the deltoid and the supra- and infra-spinatus, are decidedly atrophic, as is easily proved by weakened resistance movements. Raising the arms, especially above the shoulders, as well as adduction, are not nearly as vigorous as normal.

The weakened condition of the muscles of the shoulder girdle is well shown on attempting to lift the patient by placing the hands in the axilla. "The shoulders are raised and one gets the impression as though the patient was going to slip through" (Osler). The clavicular part of the sternomastoid is almost gone and the sternal part is much reduced. The lower part of the pectoralis major is also much atrophied. The muscles of the forearms and hands appear normal. The weakness of the back muscles is shown by the beginning lordosis. The buttocks are rather flabby and there is perhaps a slight decrease in the power of the quadriceps extensor of the thigh, tested by lowering elevated leg against resistance, while the patient is lying down. He also states that it makes him very tired to go upstairs toward the end of the day. Otherwise the thighs and legs are normal as to size, development, and power. Mitral systolic murmur at apex; apex in nipple line at fifth interspace.

In this case, while the shoulder-girdle and upper arms are the parts chiefly involved, indicating that it is of the scapulo-humeral type, still with the weakness of the facial muscles, it might be classed as a facio-scapulo-humeral type.

This case is of interest on account of the variety of the condition which it illustrates—namely, the occurrence of atrophic and hypertrophic forms in the same family. As a rule, the two atrophic forms remain distinct in the families in which they occur. These transitional cases also prove conclusively that the different types are nothing more than variations of the same disease.

This case is also of interest on account of being observed at an earlier stage than we usually see these cases, the patient not having as yet complained of any disability owing to muscular weakness. It is probable that the gradual muscular

failure dates back several years. As in the atrophic types, the disease usually takes years to make any marked progress, unless some acute illness occurs, when rapid advance takes place. In fact, the usual history of these cases is that the muscular weakness was first noticed after an acute illness or injury, a short time previous, but upon examination such extensive atrophy is found that it unquestionably dates back a much longer period.

This case also brings up the question of over-exertion during the developmental period, as a factor in causing the disease in one predisposed. Friedreich lays great stress upon this and expresses the opinion that even the normal function of learning to walk is overexertion in one predisposed, and in this way explains why the affection in children appears first in the legs and back, while in later life the arms and shoulders are first involved. Schultze, in disagreeing, points out that it remains questionable whether in early life the legs are most subject to exertion, and that it certainly does not account for those cases in which walking or standing is impossible from the start. However, that this theory has some value, is shown by the fact that members of the working classes are especially affected by the shoulder-girdle type of atrophy. In my case, overexertion might seem to have had something to do with bringing on the disease, as when seen five years previously, before starting work, he was apparently normal.

This case also illustrates the fact that was pointed out several years ago by Dr. Wharton Sinkler in a paper on family diseases, from a series of cases at the Orthopædic Hospital—namely, that in a large percentage of the cases the parents showed a syphilitic, tuberculous, alcoholic or other degenerative history. This is well shown in my case, where we have tuberculosis on the mother's side and syphilis on the father's.

THE TREATMENT OF FRACTURES OF THE FORE-
ARM, WITH NOTES OF THE END RESULTS
OF FIFTY-TWO CASES TREATED
WITHOUT OPERATION¹

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NEARLY four years ago, in a paper discussing the end results of 61 cases of fracture of the femur, treated without operation, which was presented before this Academy,² it was stated that it seemed incumbent on those surgeons who advocated operative treatment as a matter of routine in cases of recent fracture "either to demonstrate the evil results which they regard as a necessary consequence of accepted" (that is, non-operative) "methods, or to bring forward proof that by operation still better results can be obtained, and without unjustifiable risk to the patient." It was further stated: "The advocates of operative treatment, in short, should either be able to show that the methods they propose will not increase the immediate mortality and will greatly diminish or altogether prevent the unfavorable results of conservative treatment; or, failing this, they should at least convince conservative surgeons that the functional results of the accepted forms of treatment are such as can no longer be tolerated."

¹ Read before the Philadelphia Academy of Surgery, February 5, 1912. Reprinted from the American Journal of the Medical Sciences, June, 1912, No. 6, vol. cxliii, p. 843.

² Ashhurst and Newell, Annals of Surgery, 1908, ii, 748.

To our knowledge no series of cases has been published, of fractures of any long bone in the body, demonstrating either the inadequacy of conservative measures or the superiority and equal safety of operative treatment. It is to show that a fairly large series of cases of fracture of the forearm, involving both bones *in some part of their shafts*, may be treated with satisfactory results without a single resort to operation, that we now present a study of these 52 cases. From this series are excluded cases of fractures involving the elbow or the wrist-joint, and cases of Colles's fracture of the radius complicated by fracture of the ulnar styloid. Fractures of the forearm, thus limited to injuries of the shafts of the ulna and radius, do not form a large proportion of the cases of fracture seen, but they have been selected for this analysis because, next to those of the femur or leg bones, they are at present the type most often (and we believe usually quite unnecessarily) subjected to operation.

These patients were treated in the services of one of the writers at the Episcopal Hospital and at the Children's Hospital. In almost every case the patients first apply for treatment out of dispensary hours, soon after the accident which produced the fracture. They are dressed then by the surgical interne on duty in the receiving ward at the time, skiagraphs are made, and the patients are referred to the out-patient department for further treatment. Although in the very busy dispensary services in which they are treated it is not always practicable, our aim always has been to examine and dress with our own hands *all the recent fractures*, and to continue to dress them with our own hands *until union is fairly firm*. We have never relegated any cases of fracture to the care of orderlies or nurses, nor have we ever turned them over to the internes until we have ascertained by repeated and persistent personal instruction and supervision that the particular interne on duty was capable of applying the dressings in a satisfactory manner. In the treatment of fractures, as in many other important depart-

ments of surgery, one must remember that "eternal vigilance is the price of safety."

REDUCTION. "Reducing" a fracture is a relative term, since comparatively few broken bones can be accurately restored to their original form; and in the case of *shafts of long bones* it is not always necessary that reduction should be accurate. Nevertheless, the aim must be to secure as accurate reduction as possible, and in the case of fractures near joints (especially the elbow) accurate reduction is extremely important; but in the middle of the shaft of a long bone it is sufficient to secure *firm bony union*, with *no appreciable shortening*, and with *preservation of the normal axis of the limb*. For the first and second results to be obtained it is necessary for the fragments to be in contact "end-on," not only by lateral contact; and for the lateral displacement not to exceed two-thirds of the diameter of the bone.

Anesthesia rarely will be necessary in reducing a fracture of the forearm if the surgeon takes advantage of the relaxation of the muscles which may be secured by position of the limb. *Full supination of the forearm* is the position preferred, with the elbow flexed to a right angle. Correct replacement of the ulnar fracture usually can be determined clinically, as this bone is subcutaneous; but the radius is buried among so many muscles that a skiagraph frequently is necessary to ascertain the position of the fragments if the fracture is above the middle of the bone.

The forearm is then dressed *in full supination*¹ between two straight splints, specially cut to fit each individual patient.

¹ According to Malgaigne (Fractures, Paris, 1847, p. 591) this position was condemned by Hippocrates, though used by his contemporaries. Its advantages were pointed out by Paré, who was opposed to the semi-prone position until he learned that the latter had been approved by Hippocrates, whereupon Paré resumed the use of the semiprone position. Malgaigne thought he was himself the first of modern surgeons to return to the use of full supination, advised in his *Anatomie Chirurgicale* (1838), until he learned that Lonsdale (London Med. Gaz., 1832, ix, 910) had preceded him.

They should be a little wider than the forearm, so as to prevent crowding the bones together laterally, but not so wide as to permit rotation of the forearm within the splints. The palmar splint extends from the bend of the elbow to the tips of the fingers, while the dorsal splint extends from the olecranon to the wrist. These splints should be smoothly but thickly padded with raw cotton. A longitudinal pad placed between the bones, in an effort to wedge them apart, is not only useless but harmful. Extra compresses, however, may well be placed over any of the fragments that tend to project. The splints are then strapped snugly around the forearm by bands of adhesive plaster at the wrist and below the elbow, and are held securely in place by a roller bandage. A large "handkerchief" or "triangular" sling is applied, supporting the forearm throughout its length, and the forearm is carried against the chest, but always in full supination. In very small children, and in adults where the seat of fracture is near the elbow, this joint is immobilized by employing an anterior angular splint, known in Philadelphia as Hartshorne's, instead of the straight palmar splint.

The position of full supination is employed not only because supination is the most difficult part of rotation to regain,¹ if once lost, and because the upper fragment of the radius usually is kept in supination by the biceps; but because it was found by one of the writers when the forearm was dressed in mid-pronation, as commonly advised now, and as formerly employed by him, that the fragments sagged by the force of gravity, and that the patients recovered not only with lost supination, but with angular deformity of both bones toward the ulnar side. If attempt is made to correct this deformity by adjusting a coaptation splint over the angular projection of the ulna, this may indeed be overcome, but the surgeon will succeed merely in forcing the ulna nearer the radius which cannot be influenced by such an appliance,

¹ The patient regains pronation by active use of the hand; very few motions require extreme supination.

and the disability as regards rotation will be increased. However, fractures in which no tendency to displacement exists, such as greenstick and subperiosteal fractures, may be treated successfully in the semi-prone position.

Often it is exceedingly difficult to keep these fractures even approximately reduced during the first week or ten days; and it is during this period that impatient surgeons are apt to urge operation as the only solution of the difficulty. But usually a little better position can be secured at each dressing, and when the ends of the bones begin to become sticky, during the second week, it will be found that deformity daily becomes less, and what looked at first (to the inexperienced) like a hopeless case, will result in a very useful arm, and one with slight or with no visible deformity. Skiagraphs are valuable and interesting, but a surgeon never should be terrified by the appearance of the forearm bones in a skia-graph into thinking that only operative treatment can give his patient a good result. If he uses the eyes in the ends of his fingers, he will secure by conservative means quite as good, and in many cases a much better result than by operation, and in a shorter time.

AFTER CARE. This involves removal of the dressing frequently enough to make sure that the soft parts are in good condition, and that reduction is maintained by the dressings employed. The surgeon never should neglect to see the patient on the day after the dressing is first applied, and to ascertain for himself that the limb is in good condition, and that the dressing is comfortable. An uncomfortable dressing always is inefficient, even if not positively harmful; but if the dressing is comfortable it is not desirable to redress the limb more than two or three times weekly at first, and less often as union progresses. As the splints and bandages are being removed for re-dressing, the patient sits facing the surgeon, and the forearm lies supine on the patient's thigh. The palmar splint is lifted carefully off without moving the forearm, and the flexor surface and sides of the forearm and

hand are gently bathed in dilute alcohol; then without rotating the forearm at all it is gently raised as a whole from the the dorsal splint, and the extensor surface is bathed similarly, correct apposition of the fragments being maintained all the time. Any undue haste or rapid movement or attempts at rotation will be painful, will evoke muscular spasm, and may cause displacement of the fragments.

We do not approve massage or mobilization in the treatment of fractures, except in so far as they are unavoidable in procuring proper care of the soft parts; and while we acknowledge the truth of the dictum of Lucas-Championnière that "a certain amount of motion between the fragments encourages the formation of callus," we are firmly of the opinion that even the most careful immobilization by splints allows, and proper care of the soft parts, as above indicated, provides that "certain amount" of motion which is desirable; and that any surgeon who attempts more, in the vain idea that he is following modern teaching, will succeed either in stirring up such an amount of callus (especially in children) as to cause deformity and injurious pressure on the soft parts, or (in most adults) will leave his patient with an ununited fracture.

When the ends of the bones become "sticky" and no tendency to displacement exists, the surgeon may then begin to make very limited degrees of passive motion in the neighboring joints at each dressing, meanwhile maintaining support at the seat of fracture. Under no circumstances should the passive motion cause pain. When union is firm enough for all external support to be discontinued, function usually will be more comfortably and quickly recovered by active movements by the patient himself, than by further attempts at passive motion; and if a fracture has been treated properly in the first place, massage very rarely will be necessary to accelerate the cure.

OPERATIVE TREATMENT OF SIMPLE FRACTURES. We believe there are only two indications for the "open method" of treat-

ing simple fractures: (1) If the fracture cannot be properly reduced without operation, and (2) if proper reduction cannot be maintained without direct fixation of the fragments.

1. *When Proper Reduction is Impossible.* Impossibility is here a relative term, since what is impossible for one surgeon may not be so for another; and the qualification "proper" reduction is employed because we do not wish to imply that operation is indicated whenever accurate, exact, perfect, anatomical replacement is impossible, but only when such degree of reduction, as has been described in a previous paragraph as requisite for proper function, cannot be secured without open operation. Less perfect reduction is requisite in children than in adults, since in the former compensation is more rapidly established; and oblique fractures require less close and accurate apposition of their ends than do transverse fractures.

2. *When Subsequent Displacement Cannot be Prevented.* This also is a relative condition, depending on the skill of the surgeon in devising and applying efficient retentive apparatus, and upon the extent to which displacement occurs.

After operation the process of union often is slower than it would have been if no operation had been employed; and in a fair proportion of cases operated on by the average surgeon a mild degree of infection occurs, and only fibrous union results.

STATISTICS. These cases form a continuous series, absolutely unselected, running through a period of ten years. Of 66 patients treated, the end results are known in 52 cases; 43 of the fractures occurred in males, and only 9 in females. None of the female patients was older than fifteen years. Of the males, 31 were less than fifteen years of age, and 12 were older. The youngest patient (a girl) was seventeen months old, and the oldest (a man) was fifty-seven years. The right and left arms were affected nearly with equal frequency.

For estimating the power of rotation, a special instrument (Fig. 46) was devised, and was constructed for us by D. W.

Kolbe Co. For establishing a standard, the average normal rotation was ascertained by testing one hundred normal forearms of fifty persons (25 male, 25 female). The instrument consists of an indicator, kept vertical by gravity, and centered on a circular protractor. This protractor is attached to an upright board which itself is fastened at right angles to a horizontal board. The horizontal board is strapped on the flexor surface of the wrist, the forearm being in full supination. In this position the indicator points to 0 degrees. If still further supination is possible, this is recorded as a *minus* quantity, of say 5, 10, or 15 degrees. As the forearm is rotated into pronation the protractor also rotates, but the indicator remains vertical and passes over the rotating scale from 0 degree up to 135 degrees or 140 degrees or more, according to the extent of pronation possible (Fig. 47). During this examination, it is needless to say, the patient's humerus should be kept immovably applied to the side of the thorax, and no deviation of the body from the vertical should be permitted.

NORMAL ROTATION. Our examination of 100 normal forearms gave the following results:

Ages varied from fourteen to seventy-seven years; average age, twenty-nine years.

Supination

Greatest	. . .	—45.00 degrees	Least	. . .	15.00 degrees
Average.	. . .	—11.72 degrees	{ In males	. . .	— 7.42 degrees
			{ In females	. . .	—16.02 degrees
Average of right forearm —7.24 degrees; of left —16.2 degrees					

Pronation

Greatest	. . .	180.00 degrees	Least	. . .	120.00 degrees
Average.	. . .	147.77 degrees	{ In males	. . .	148.24 degrees
			{ In females	. . .	147.30 degrees
Average of right forearm, 147.24 degrees; of left, 148.00 degrees.					

Greatest range of rotation in any one patient, from —45 degrees to 155 degrees, or 200 degrees.

Least range of rotation in any one patient from 0 degrees to 122 degrees, or 122 degrees.

Average rotation from —11.72 degrees to 147.77 degrees, or 159.49 degrees.

RESULTS. In studying the end results of these 52 cases, it may first be stated what was not secured: there was no case of gangrene of the soft parts or of necrosis of the bones; there was no case of ischemic contracture; none of nerve lesion; none of ununited fracture; none of conspicuous deformity. There was one case of delayed union (Case 39), but this patient cured himself, by returning to his work as a blacksmith at the end of ten weeks. There were several cases in which some thickening or irregularity could be felt at the site of fracture; but none in which these were appreciable at a glance. There was no case of disability, even slight. And these results were obtained without excluding 9 more or less complicated cases, as follows: 1 case of badly comminuted fracture (Case 39); 1 case of fracture of both bones of both forearms (Cases 2 and 3); 3 cases of multiple fractures of the upper extremity (Cases 1, 25, 26) involving both the humerus and the bones of the forearm, two of which were compound comminuted fractures; as well as 3 other cases of compound fracture (Cases 7, 32, 47). None of the cases of compound fracture, however, was such as to require operation on account of the condition of the soft parts.

END RESULTS OF FIFTY-TWO CASES OF FRACTURE OF BOTH BONES OF THE FOREARM

Character of fracture	Class I. Perfect result.	Class II. Slight deformity.	Class III. Rotation limited.
Greenstick	10	1	0
Simple complete	22	8	4
Simple comminuted	0	0	1
Compound	1	1	1
Compound comminuted	1	0	2
Total	34 (65.4%)	10 (19.2%)	8 (15.4%)

The end results may be seen at a glance in the above table. Under the heading "perfect result" we include only such cases as have recovered without palpable deformity, and with preservation of perfect function. If, in spite of



FIG. 46.—Pronometer, or instrument for measuring the degree of pronation and supination. Indicator points to zero degrees in supine position.



FIG. 47.—Pronometer. | Indicator points to 90 degrees when forearm is in "midpronation."



FIG. 48.—Case I. Five years after compound fracture of both bones of forearm, and fracture of humerus. No deformity. Perfect function.

preservation of perfect function, there is palpable deformity, as in Cases 2 and 3, the patients are recorded in the second column, as "slight deformity, but perfect function." If there is limitation of function the cases are placed in the third column; in none was there any limitation of function except in rotation; and in none was there any disability.

The skiagraphs of end results, which we have chosen for reproduction here, are those of cases in which the prognosis seemed least favorable, and represent, therefore, much worse than the average skiagraphic results. As far as cosmetic results are concerned, there was no visible deformity in any of these cases except in Cases 2, 3, 26, 39, 48, and 51; and in these patients a mere glance at the bared arm will not detect any deformity.

In Class I we have included Cases 1, 4, 5, 6, 8, 10, 11, 12, 14, 16, 17, 18, 19, 22, 23, 24, 25, 27, 28, 29, 31, 34, 35, 36, 37, 38, 41, 42, 43, 44, 45, 46, 50, and 52.

In Class II are included Cases 2, 3, 20, 21, 30, 33, 40, 47, 48, and 49.

In Class III are included Cases 7, 9, 13, 15, 26, 32, 39, and 51.

We are greatly indebted to Dr. Thos. S. Stewart, radio-grapher to the Episcopal Hospital, and to his assistant, Dr. A. R. Wilkinson, for the interest they have taken in providing us with numerous skiagraphs for the purpose of this study.

ABSTRACTS OF CASE HISTORIES

CASE 1.—Michael C., aged fifteen years. May 6, 1902. Episcopal Hospital. Multiple fractures of right upper extremity (surgical neck of humerus, both bones of forearm, compound of ulna). Recorded as Case I in a paper on "Multiple Fractures" (Ashhurst, *Annals of Surgery*, 1907, ii, 263). Full supination. Examined February 20, 1907. No visible or palpable deformity anywhere. Does heavy laboring work, and would not know arm ever had been injured. Perfect result. Class I. (Fig. 48).

CASES 2 and 3.—Augustus F., aged thirty-five years. March 16, 1903. Episcopal Hospital. Fracture of both bones of both

forearms, sixteen days previously; has been dressed on internal angular and short dorsal splints, in position of mid-pronation. Arms gave constant pain. No attempt at union, because fragments were not in contact and were not immobilized. By putting forearms in full supination better position was secured, but complete reduction was impossible. However, pain was immediately and absolutely relieved. Two weeks later, good union. Examined October 28, 1911. Says he never knows arms were broken; was out of work only fourteen weeks in all, and has worked as machinist ever since with perfect function. There is no visible deformity, but the displaced fragments are still palpable. Supination in both forearms complete; pronation on right to 135 degrees, and on left to 130 degrees. Without seeing the skiagraphs made recently (Figs. 49 and 50), the results might be considered perfect. Class II.

CASE 4.—Harry H., aged ten years. September 1, 1903. Episcopal Hospital. Right. Treated in supination. Examined October 28, 1911. Full supination, pronation to 165. Class I.

CASE 5.—Phoebe G., aged two years. October 15, 1903. Episcopal Hospital. Right, greenstick. Examined October 28, 1911. Full supination, pronation to 160. Class I.

CASE 6.—Adolph W., aged ten years. October 19, 1903. Episcopal Hospital. Right. Examined, January 28, 1912. Broken arm, supination —20 degrees, pronation 130 degrees; normal arm, supination —15 degrees, pronation 135 degrees. Class I.

CASE 7.—William T. S., aged forty years. January 18, 1904. Episcopal Hospital. Right, simple of radius, compound comminuted of ulna. Examined five weeks later. Function good, but some limitation of supination. Class III.

CASE 8.—Fred. S., aged fifteen years. January 18, 1904. Episcopal Hospital. Dressed in full supination. Examined March, 1904. Class I.

CASE 9.—Thomas M., aged twenty-five years. January 19, 1904. Episcopal Hospital. Left, above middle. Great and persistent overlapping, with projection of upper fragments of radius and ulna on extensor surface. Dressed in mid-pronation, on internal angular and short dorsal splint. Examined March 12, 1904. Very slight deformity; no supination beyond mid-position. Class III.

CASE 10.—George H., aged twenty-eight years. January 23, 1904. Episcopal Hospital. Left, middle third. Dressed in mid-pronation. Examined, March 12, 1904. Class I.

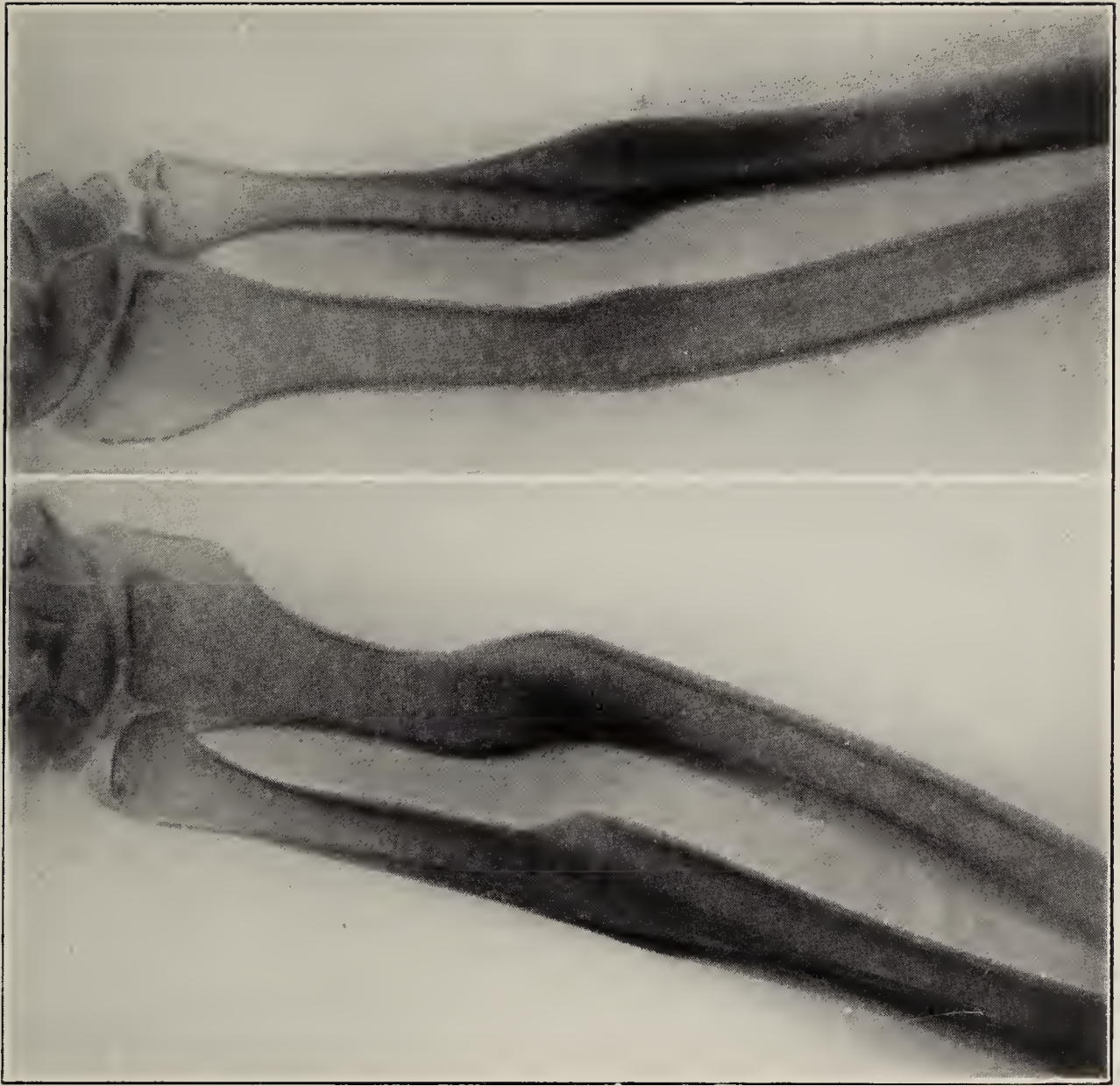


FIG. 49.—Cases II and III. Anteroposterior views of both forearms, eight years after injury. Function perfect.

CASE 11.—Emma L., aged two years. January 31, 1904. Episcopal Hospital. Greenstick. Examined March 3, 1904. Class I.

CASE 12.—William A., aged twelve years. April 12, 1904. Episcopal Hospital. Left, treated in mid-pronation. Examined October 28, 1911. Supination full, pronation to 135 degrees. Class I.

CASE 13.—Fred. H., aged sixteen years. April 15, 1904. Episcopal Hospital. Right, dressed in mid-pronation. Had refracture of same forearm in November, 1904. Examined January 27, 1912. No deformity, no disability. Right: supination, 25 degrees; pronation, 120 degrees. Left: supination, 20 degrees; pronation, 145 degrees. Class III.

CASE 14.—Albert S., aged thirteen years. September 1, 1904. Episcopal Hospital. Greenstick. Treated in mid-pronation. Examined October, 1904. Class I.

CASE 15.—James M., aged eight years. September 8, 1904. Episcopal Hospital. Left lower third. Very great cellulitis. Treated in mid-pronation. Examined October, 1904. No deformity, but little supination beyond mid-position. Class III.

CASE 16.—Thomas McG., aged fourteen years. December 7, 1905. Episcopal Hospital. Right; of radius above insertion of pronator teres, and greenstick of ulna, same level. Treated in full supination. Examined December 29, 1905. No deformity, full supination, pronation good. Class I.

CASE 17.—Anna D., aged fourteen years. December 23, 1905. Episcopal Hospital. Right. Treated in full supination. Examined January 15, 1906. Class I.

CASE 18.—John F., aged thirteen years. December 29, 1905. Episcopal Hospital. Middle third. Treated in full supination. Examined October 28, 1911. Full supination, pronation 165 degrees. Class I.

CASE 19.—Juliette J., aged eight years. July 3, 1906. Children's Hospital. Greenstick, middle third. Treated in full supination. Examined August 2, 1906. Class I.

CASE 20.—Carrie C., aged six years. August 2, 1906. Children's Hospital. Injury two weeks ago at Atlantic City, and dressed in mid-pronation. On admission today, fracture at junction of middle and lower thirds of right forearm; no union, fair position; but bowing of ulna to extensor surface. Treated in full supination. August 21, union good, no deformity; full supination and pronation. Examined August 28, 1906. Upper fragment of ulna displaced slightly posteriorly; functions perfect. Class II.

CASE 21.—John S., aged twelve years. August 6, 1906. Children's Hospital. Left, junction of middle and lower thirds. Fell from tree; was stunned. Upper fragment of ulna projects beneath skin of flexor surface; both lower fragments displaced toward extensor and radial surfaces. Treated in full supination. August 10. Deformity persists; pad over upper fragment of ulna. August 14. Position better; growing firmer. August 21. Position fair; bones firm. Examined September 5, 1906. Little deformity, no disability; supination and pronation complete. Class II.

CASE 22.—William G., aged seven years. November 3, 1906. Episcopal Hospital. Left, greenstick, middle third. Dressed in full supination. Then patient visited another dispensary, and when splint applied there fell off of itself, he returned to Episcopal Hospital, November 19, with marked dorsal bowing of both bones. Bones were re-fractured, deformity reduced, and forearm dressed in full supination. Examined December 10, 1906. No visible deformity, but a little callus palpable over radius. Full supination and pronation. Class I.

CASE 23.—Albert S., aged eleven years. November 15, 1906. Episcopal Hospital. Right, complete of radius, greenstick of ulna. Dressed in full supination. Examined December 12, 1906. Class I.

CASE 24.—Hugh F., aged fourteen years. November 21, 1906. Episcopal Hospital. Left, above wrist. Full supination. Examined October 28, 1911. Supination complete, pronation to 180 degrees. Class I.

CASE 25.—Andrew M., aged fourteen years. December 22, 1906. Episcopal Hospital. Left; compound comminuted fracture of both bones of left forearm with compound comminuted fracture of left humerus. (Reported as Case VI in paper on "Multiple Fractures," in *Annals of Surgery*, 1907, ii, 263.) Treated in ward for nine days, then in dispensary. Forearm dressed in full supination. Fig. 51 is from a photograph taken three months after the accident. Examined October 28, 1911. Supination and pronation complete. No deformity. Figs. 52 and 53 are from skiagraphs made in January, 1912. Class I.

CASE 26.—Henry D. E., aged fifty-seven years. November 26, 1906. Episcopal Hospital. Left. Compound comminuted fracture of both bones of left forearm, with comminuted fracture of left humerus. (Reported as Case V., in paper on "Multiple Fractures," in *Annals of Surgery*, 1907, ii, 263.) Treated with forearm in full supination. Examined January 21, 1912. Was

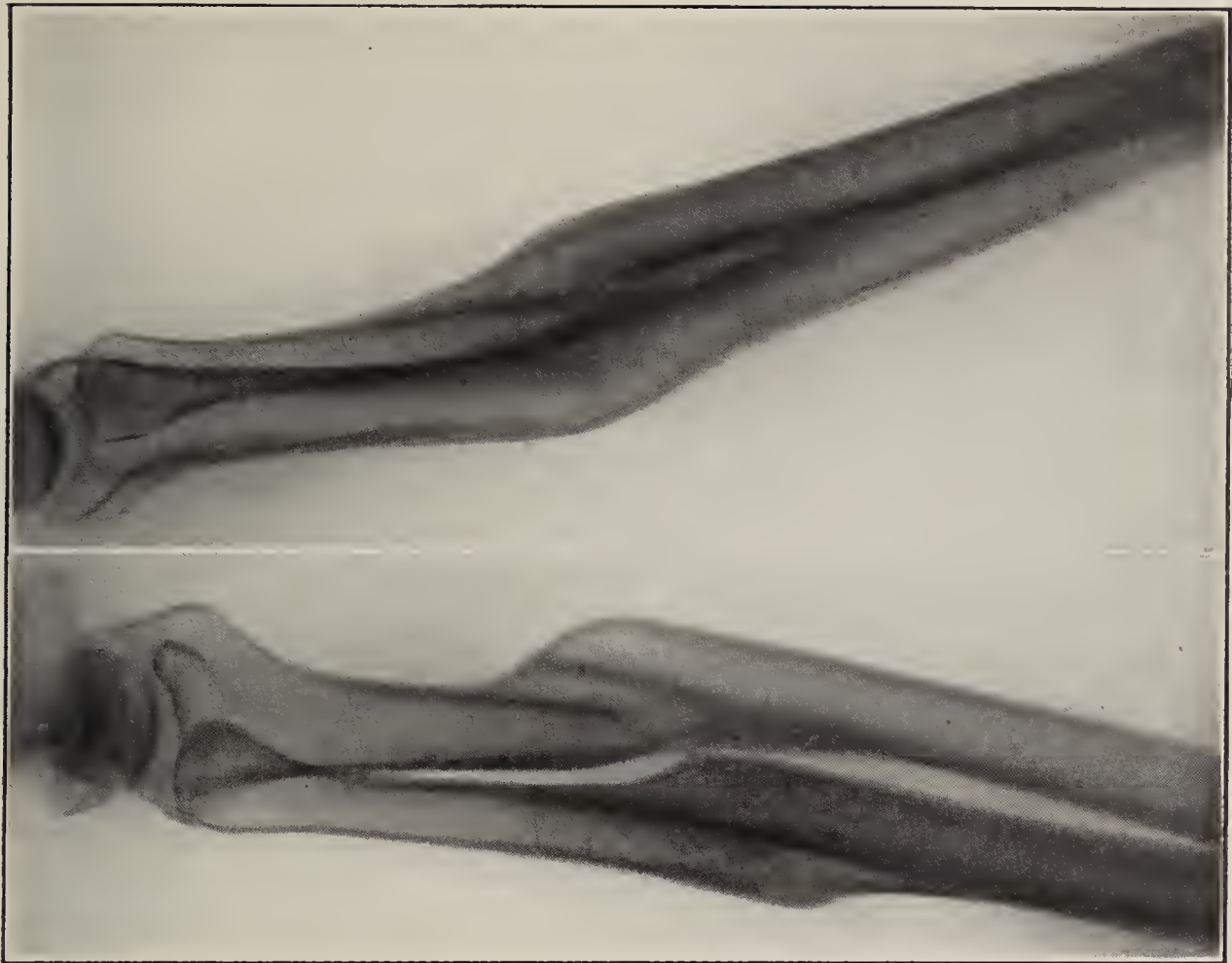


FIG. 50.—Cases II and III. Lateral view of both forearms eight years after injury. Function perfect. Patient first came under the writer's care sixteen days after injury.



FIG. 51.—Case XXV. Compound comminuted fracture of both bones of left forearm, with compound comminuted fracture of left humerus. No deformity. Perfect function. See Figs. 52 and 53.



FIG. 52.—Case XXV. Lateral view of forearm five years after compound comminuted fracture of both bones.

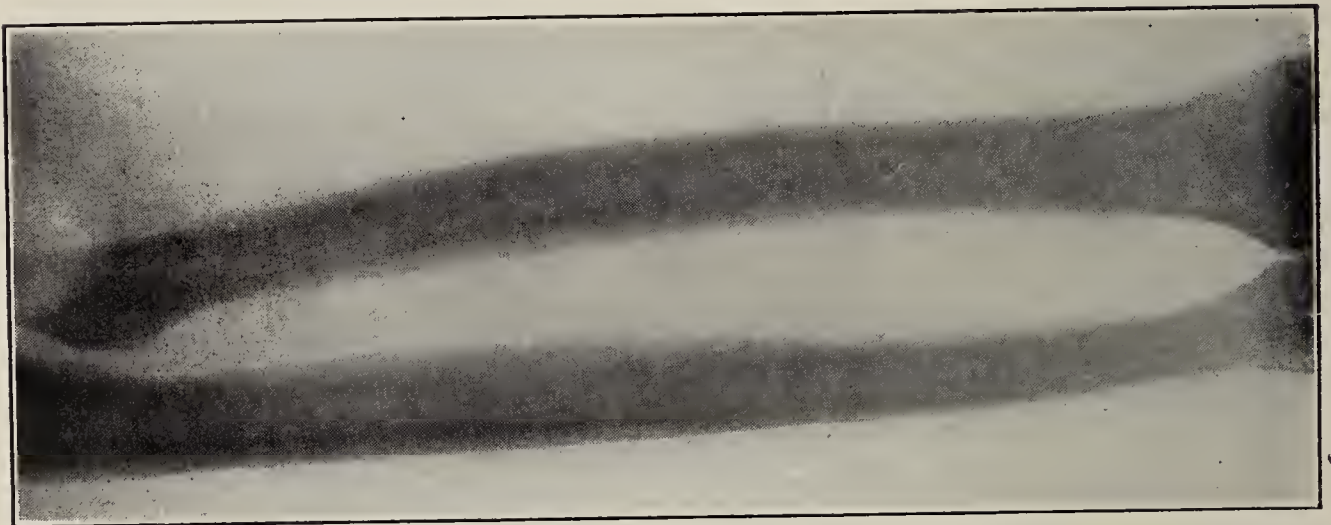


FIG. 53.—Case XXV. Anteroposterior view of forearm, five years after compound comminuted fracture of both bones.

out of work (saw-maker) for four months. Complete function not regained for one year. Since then has experienced no disability whatever. There is considerable deformity in forearm, bones being bowed to radial side. Left forearm: supination, 0 degrees; pronation, to 85 degrees. Right forearm: supination, 0 degrees; pronation, to 155 degrees. Class III.

CASE 27.—Albert B., aged three years. March 5, 1907. Episcopal Hospital. Right, greenstick, two weeks old; bones bowed to extensor surface, some callus; inability to supinate completely. Refractured, and dressed in full supination. Examined April 4, 1907. No deformity, supination and pronation complete. Class I.

CASE 28.—Harriet B., aged two years. March 29, 1907. Episcopal Hospital. Left, greenstick. Full supination. Examined April 26, 1907. Supination and pronation complete. Class I.

CASE 29.—Charles G., aged fourteen years. January 9, 1908. Episcopal Hospital. Left, full supination. Examined February 13, 1908. Class I.

CASE 30.—Harry W., aged thirty months. July 8, 1907. Children's Hospital. Left, greenstick. One month's duration. Very marked angulation just above wrist, about 135 degrees, angle being open on extensor surface. Refractured (ether) and dressed in full supination. July 15, no union yet. July 20, some union. Examined, August 15, 1907. Slight radial deviation of hand, due to rachitic deformity, same as in other arm. Supination and pronation complete. Class II.

CASE 31.—George W. D., aged fourteen years. July 23, 1907. Children's Hospital. Right, lower third. Full supination. Examined August 29, 1907. Supination complete, pronation about 140 degrees. Class I.

CASE 32.—Dillman F., aged five years. June 21, 1907. Children's Hospital. Right; compound of ulna. Dressed in mid-pronation. Examined August 8, 1907. Supination not quite complete, pronation complete. Some callus over radius, and slight deformity to flexor surface. Functions perfect. Class III.

CASE 33.—Clark W. B., aged four years. July 29, 1907. Children's Hospital. Left; same forearm was broken two years ago. Dressed in full supination. Examined August 29, 1907. Both bones bowed slightly to radial aspect; rotation from full supination is good. Class II.

CASE 34.—John H., aged twenty-two months. January 13, 1908. Episcopal Hospital. Left, greenstick. Full supination. Examined February, 1908. Class I.

CASE 35.—Marcus D., aged sixteen years. April 14, 1908. Episcopal Hospital. Epiphyseal separation of radius and greenstick of ulna above styloid. Full supination. Examined October 28, 1911. Class I.

CASE 36.—Louis S., aged fourteen years. April 22, 1908. Episcopal Hospital. Left, lower fifth. Full supination. Examined June 2, 1908. Class I.

CASE 37.—Charles W. H., aged 18 months. January 4, 1909. Episcopal Hospital. Left, greenstick, Full supination. Examined March, 1909. Class I.

CASE 38.—Clara Y., aged nine years. February 10, 1909. Episcopal Hospital. Right, two inches above wrist. Dressed on Bond splint, in mid-pronation, there being no tendency to deformity. Examined October 28, 1911. Class I.

CASE 39.—Michael S., aged fifty-three years. February 25, 1909. Episcopal Hospital. Right; comminuted; was caught in machinery. Dressed in full supination; accurate reduction not secured (Fig. 54). Delayed union. After ten weeks returned to his work (blacksmith), and two months later had firm union. Examined January 20, 1912. Right forearm: supination, 15 degrees; pronation, 105 degrees. Left forearm: supination, 0 degrees; pronation, 140 degrees. There is slight palpable bony deformity, but no disability whatever (Fig. 55). Class III.

CASE 40.—Victoria K., aged seventeen months. February 16, 1910. Episcopal Hospital. Right. Dressed in mid-pronation. Examined ten weeks later. Slight extensor bowing of ulna; supination and pronation complete. Class II.

CASE 41.—Wilson McC., aged fourteen years. March 23, 1910. Episcopal Hospital. Right, lower third. Full supination. Examined, January 20, 1912. No deformity palpable. Right forearm: supination, —35 degrees; pronation, 125 degrees. Left forearm: supination, —40 degrees; pronation, 140 degrees. Class I.

CASE 42.—William E., aged fifteen years. January 18, 1911. Episcopal Hospital. Right, above wrist. Full supination. Examined October 28, 1911. Class I.

CASE 43.—Helen K., aged three years. January 28, 1911. Episcopal Hospital. Right, greenstick. Full supination. Examined October 28, 1911. Supination complete, pronation to 170 degrees. Class I.

CASE 44.—John L., aged seventeen years. August 11, 1911. Episcopal Hospital. Complete of radius, greenstick of ulna. Full supination. Examined October 28, 1911. Perfect result. Supination complete, pronation to 160 degrees. Class I.

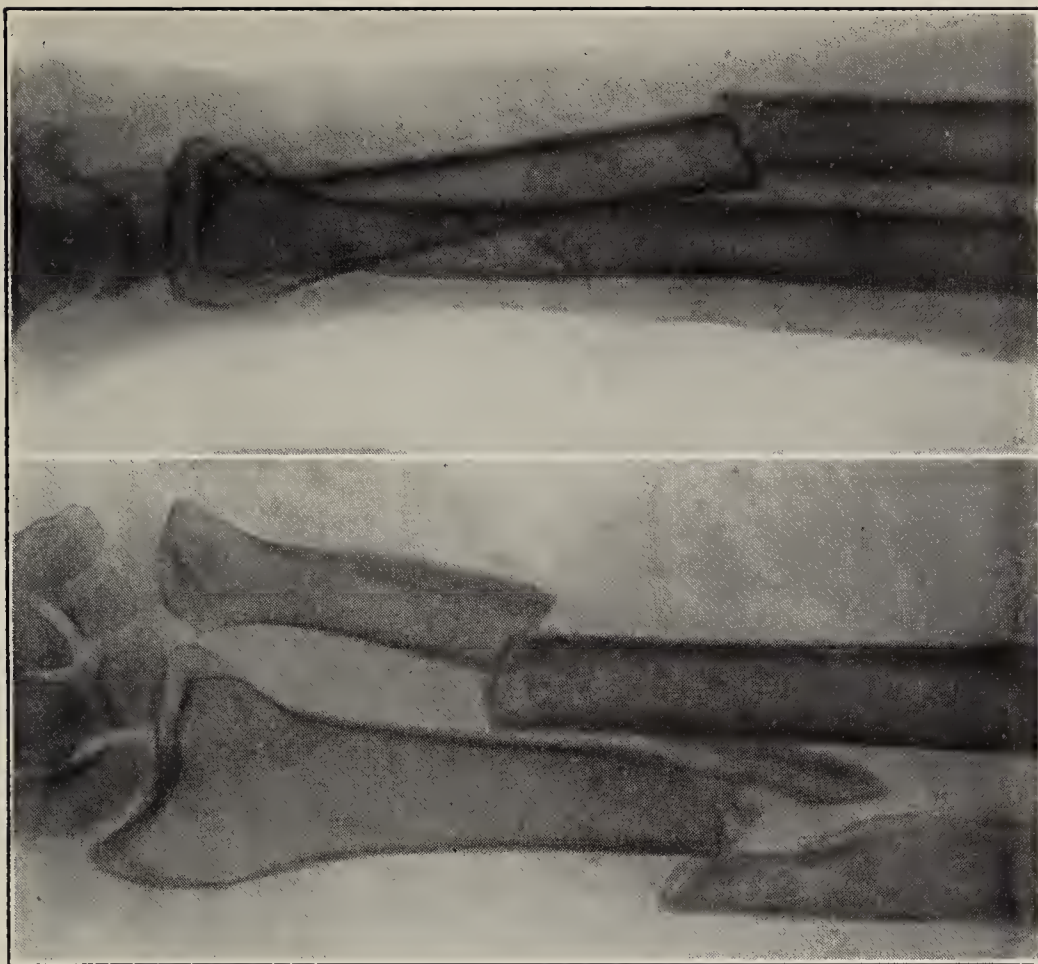


FIG. 54.—Case XXXIX. Lateral and anteroposterior views of comminuted fracture of both bones of forearm, showing best position secured. See Fig. 55.

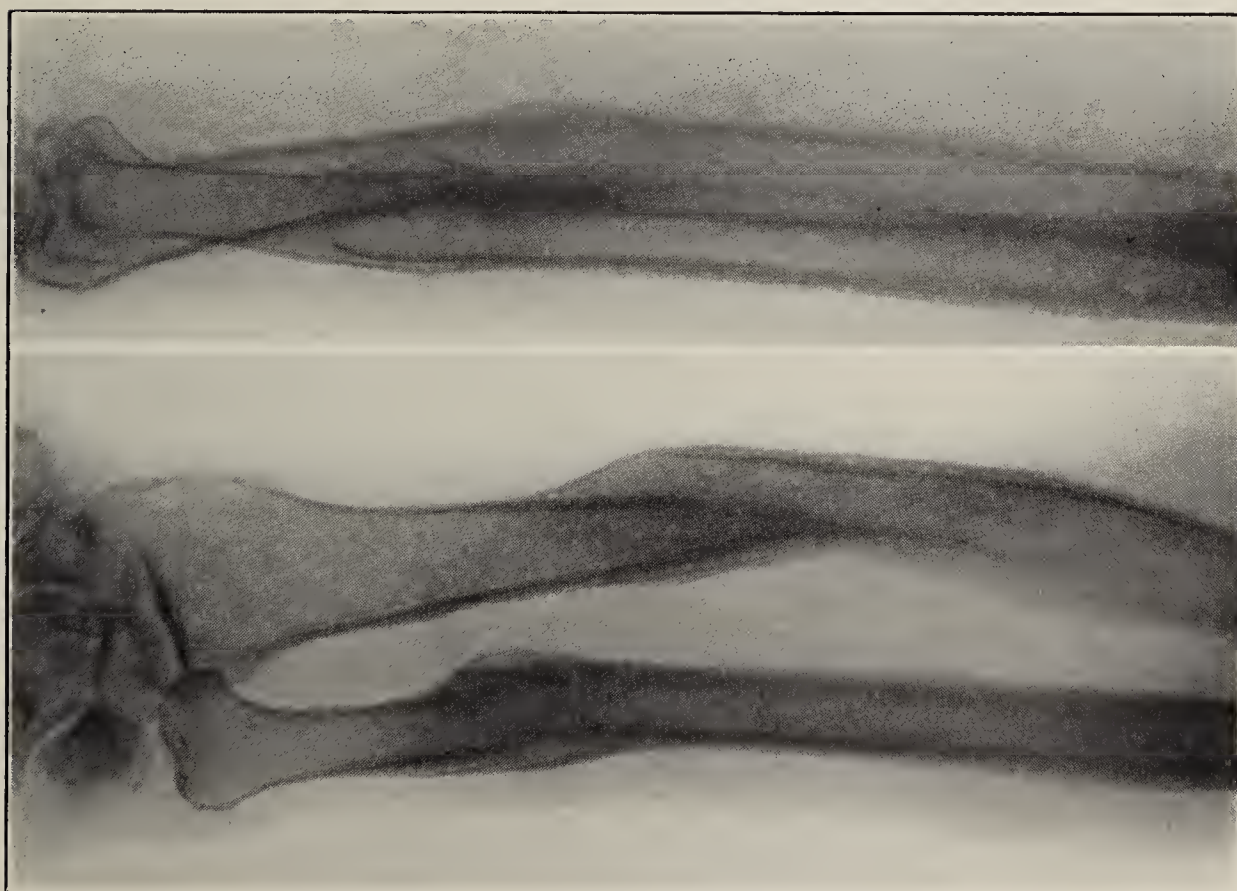


FIG. 55.—Case XXXIX. Lateral and anteroposterior views of comminuted fracture of both bones of forearm, three years after injury. There was delayed union, but patient was out of work only ten weeks in all. This is the worst result in the entire series.



FIG. 56.—Case XLVII. Compound fracture of radius and ulna, before coming under care of the writers, and while still dressed in midpronation. See Figs. 57 and 58.

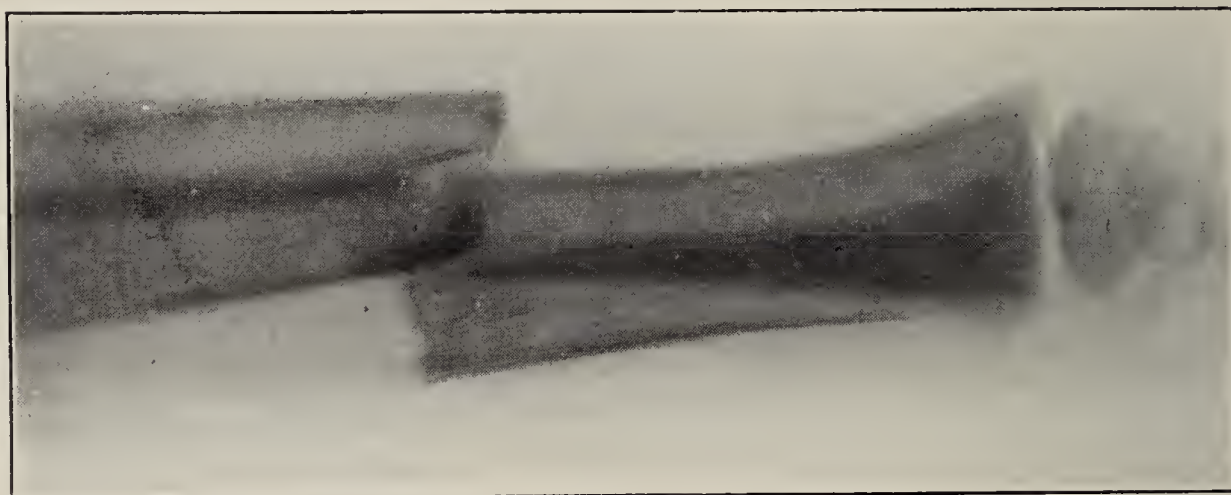


FIG. 57.—Case XLVII. Lateral view, after dressing in full supination. one week after injury.

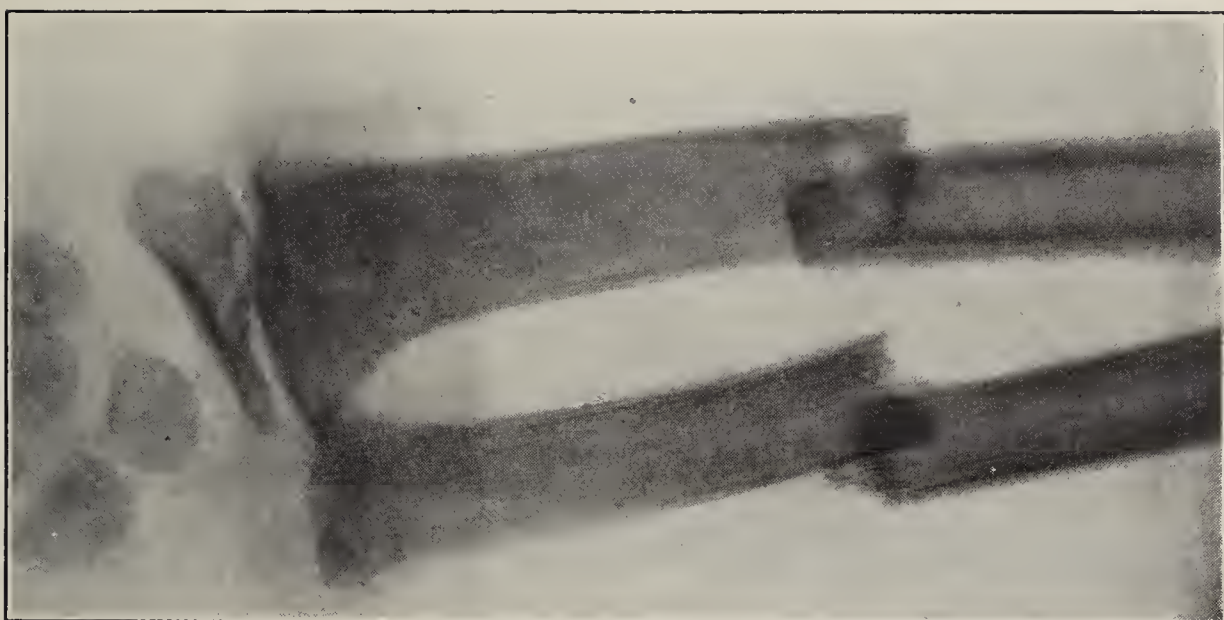


FIG. 58.—Case XLVII. Anteroposterior view after dressing in full supination.

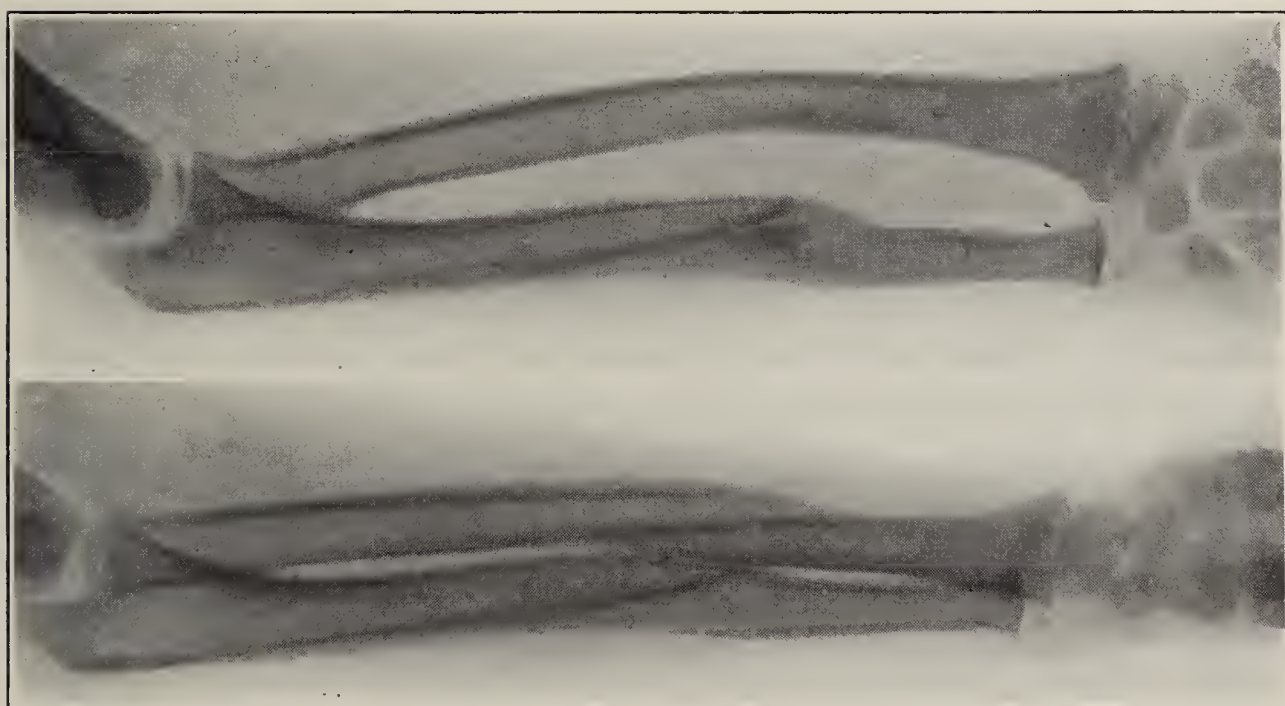


FIG. 59.—Case XLVII. Compound fracture of both bones of forearm five months after injury. Slight callus palpable. No visible deformity. Perfect function.

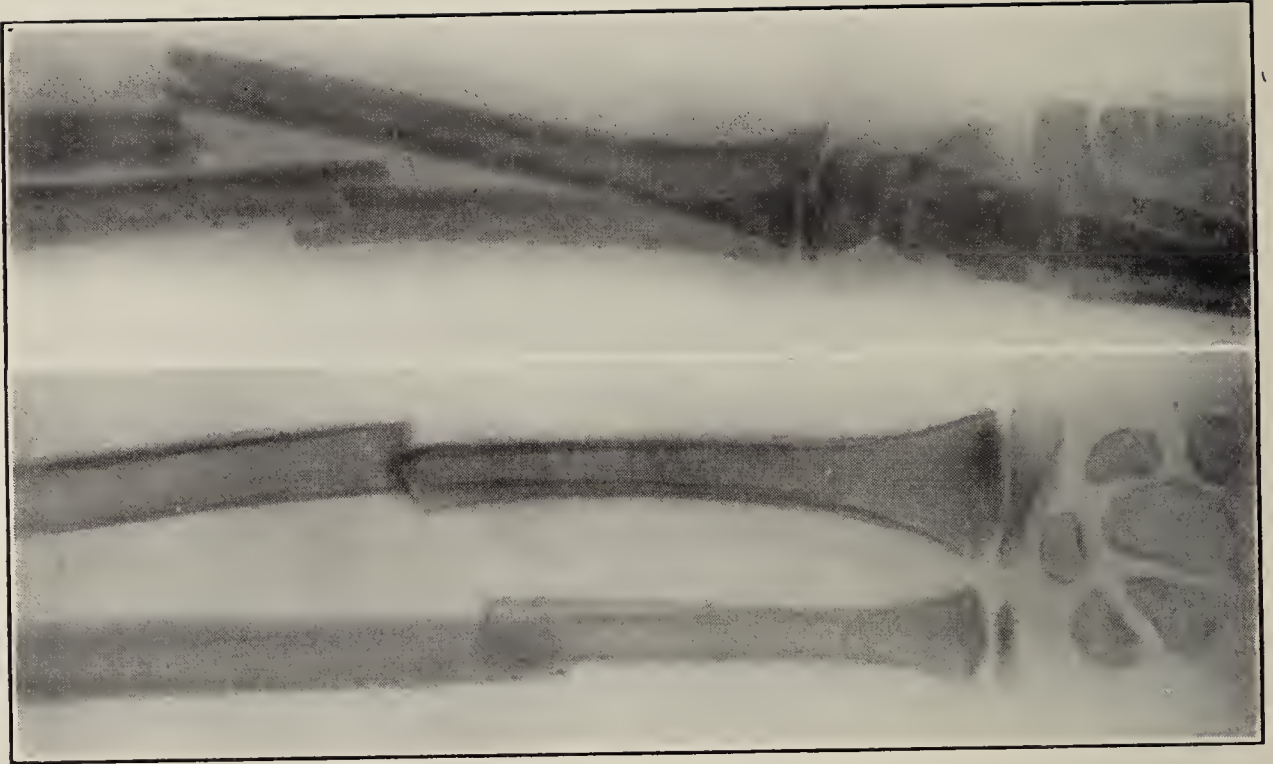


FIG. 60.—Case XLIX. Lateral and anteroposterior views after first dressing. See Fig. 61.

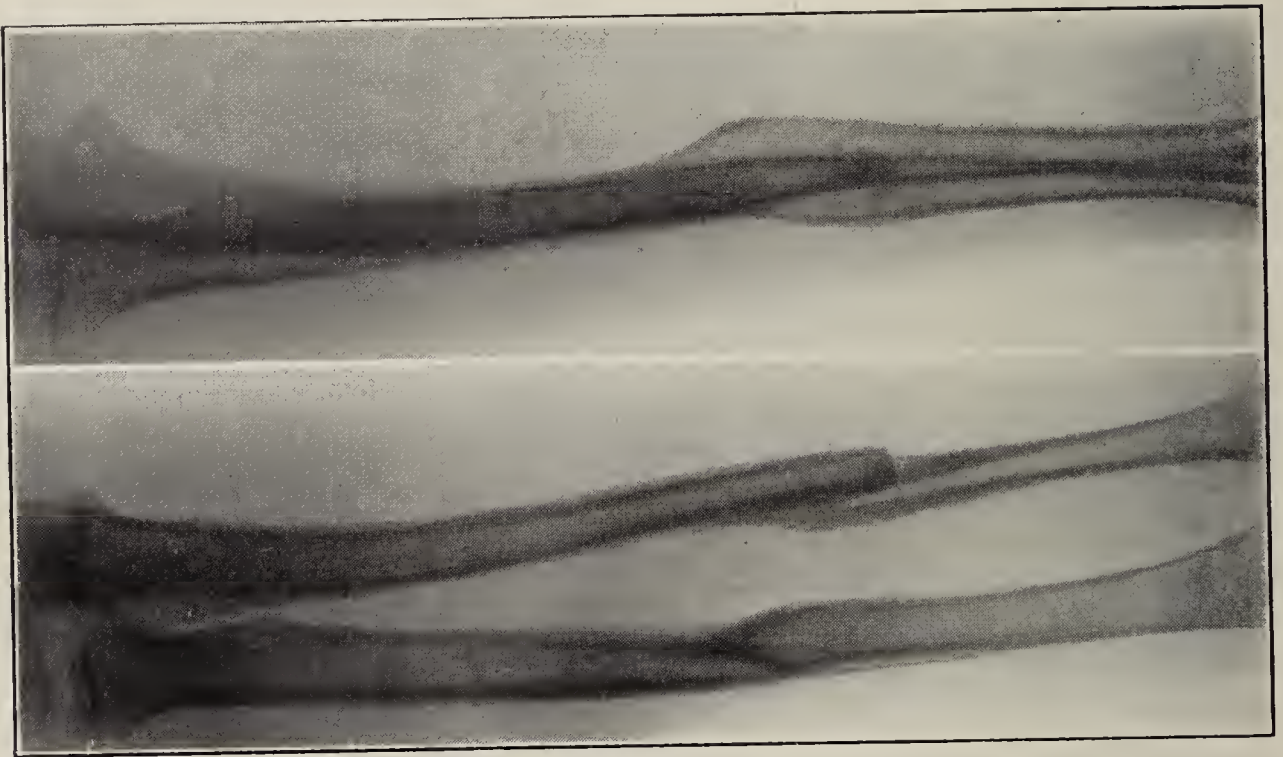


FIG. 61.—Case XLIX. Lateral and anteroposterior views four months after injury. Slight callus palpable. No visible deformity. Perfect function.

CASE 45.—George S., aged nine years. August 11, 1911. Episcopal Hospital. Middle third, left; has been dressed in mid-pronation at another hospital. Examined October 27, 1911. Supination complete, pronation to 135 degrees. Class I.

CASE 46.—Harry M., aged thirteen years. August 16, 1911. Episcopal Hospital. Right, above wrist. Full supination. Examined October 28, 1911. Supination complete, pronation to 135 degrees. Class I.

CASE 47.—Roger McB., aged nine years. August 17, 1911. Episcopal Hospital. Injury one week ago, and was referred as suitable for operation; had been dressed in mid-pronation (Fig. 56). Better position secured by dressing in full supination (Figs. 57 and 58), and no operation recommended. Compound fracture of radius and ulna, wounds on flexor surface of forearm. Examined January 20, 1912. Slight amount of callus palpable. Injured forearm: supination, —30 degrees; pronation, 130 degrees. Normal forearm: supination, —20 degrees; pronation, 135 degrees. Class II. (Fig. 59).

CASE 48.—Tony M., aged fourteen years. September 8, 1911. Episcopal Hospital. Left; injury one week ago, junction of middle and upper thirds. Had been dressed in mid-pronation. Better position secured by dressing in full supination. Examined January 21, 1912. Slight posterior bowing of both bones. Left forearm: supination, 20 degrees; pronation, 150 degrees. Right forearm: supination, —5 degrees; pronation, 140 degrees. Class II.

CASE 49.—Richard W., aged thirteen years. September 23, 1911. Episcopal Hospital. Left, middle third. Full supination. Skiagraph (Fig. 60) showed lower fragment of radius displaced to flexor surface. Examined, January 17, 1912. No visible deformity, slight callus palpable on flexor surface of radius (Fig. 61). Left forearm: supination, —15 degrees; pronation, 125 degrees. Right forearm: supination, —10 degrees; pronation, 135 degrees. Class II.

CASE 50.—Herbert T., aged fourteen years. September 26, 1911. Episcopal Hospital. Right, middle third. Dressed in full supination. Examined January 21, 1912. Some callus palpable. Right forearm: supination, —10 degrees; pronation, 155 degrees. Left forearm: supination, —10 degrees; pronation, 140 degrees. Class I.

CASE 51.—John D., aged eighteen years. October 10, 1911. Episcopal Hospital. Right, junction of middle and upper third. Full supination. Some projection of upper fragment of radius on

flexor surface. Examined January 28, 1912. Slight callus of radius. Right forearm: supination, 40 degrees; pronation, 135 degrees. Left forearm: supination, —5 degrees; pronation, 120 degrees. Class III. (This patient was under the care of the writers only seventeen days.)

CASE 52.—Stanley C., aged fourteen years. October 10, 1911. Episcopal Hospital. Left, lower third of radius, and through lower epiphysis of ulna. Full supination. Examined January 27, 1912. Left forearm: supination —15 degrees; pronation, 160 degrees. Right forearm: supination, —5 degrees; pronation, 160 degrees. Class I.

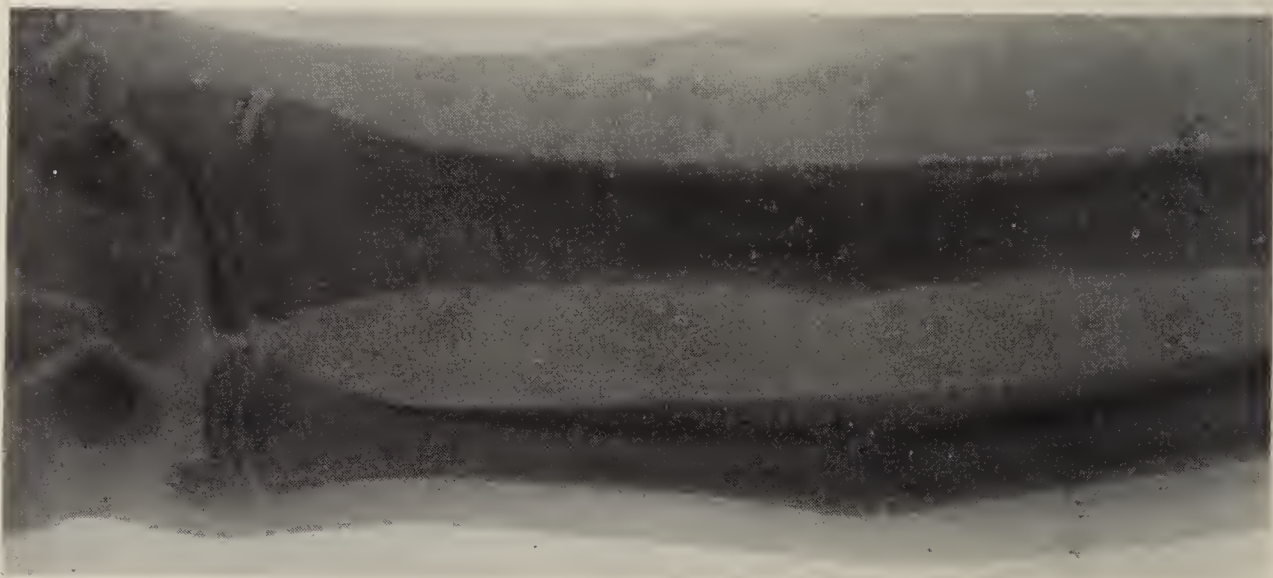


FIG. 62 *a*.—Fracture of both bones of forearm, which was dressed in mid-pronation; united with angular deformity due to effect of gravity. Anteroposterior view. Case I.

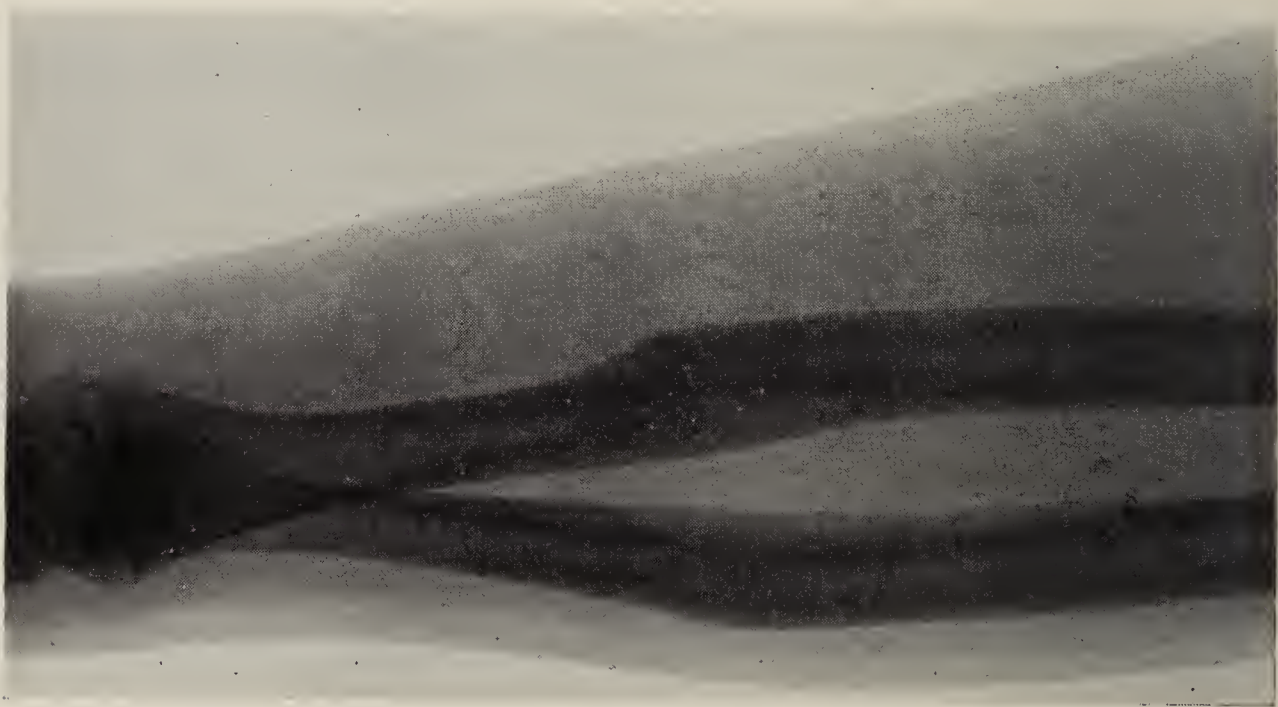


FIG. 62 *b*.—Fracture of both bones of forearm which was dressed in mid-pronation; united with deformity. Lateral view. Case I.

TREATMENT OF FRACTURE OF BOTH BONES OF THE FOREARM IN THE POSITION OF FULL SUPINATION¹

By JOHN C. SIMPSON, M.D.
RESIDENT PHYSICIAN AT THE HOSPITAL

IN the treatment of a fracture of both bones of the forearm, reduction should be attempted by direct traction, grasping the hand and holding the upper arm firmly. Traction made by holding the forearm above the wrist tends to press the two lower fragments together, thus reducing the width of the interosseous space. Complete reduction is not absolutely necessary for good functional result.

After reduction, it is the teaching of practically every text-book that the arm should be splinted and maintained in mid-pronation, the idea being that in this position the interosseous space is at its greatest width, and that thus there is less danger of the callus at the seat of the fractures obliterating this space, and so greatly interfering with the movements of pronation and supination, or entirely prohibiting them.

It is evident, in a forearm held in this position with splints, anteriorly and posteriorly, with no support in the lines of the force of gravity, that as soon as the dressings become loosened, gravity tends to pull the fragments down, as the arm is not supported on the under side. This gives a bowing of the

¹ Read before the Episcopal Hospital Clinical Society, November 18 1912.

fragments downward, which usually causes some loss of the ability to supinate, and gives a permanent deformity.

To prevent the possibility of this deformity, all fractures of the forearm treated in Dr. Ashhurst's service are dressed in full supination, using splints just wide enough to prevent crowding of the fragments together, but not wide enough to permit of rotation of the arm within the splints. The posterior splint then acts as a support to the fragments in the line of gravity and obviously prevents bowing toward the ulnar side. The anterior splint acts as a further support, and prevents the dressings from crowding the bones together. A further point in favor of treating these fractures in full supination is that all the active movements made by the patient after recovery tend to restore to him the power of full pronation, while few if any natural movements encourage the return of full supination. It is the most difficult movement to regain if once lost.

I wish to report two typical cases that came under our care in the surgical dispensary last summer, one treated in the position of mid-pronation, the other in full supination.

CASE I.—A boy received a fracture of both bones of the right forearm, by a fall, about the middle of last July. He was dressed in the receiving ward in mid-pronation and was referred to the dispensary. Throughout the entire course of his treatment his arm was held in mid-pronation. After having been under treatment for one week he left the city and remained away for one week. During this week his arm was not dressed and the dressings became loose. On his return, when he first entered Dr. Ashhurst's service, we noticed a marked bowing downward of the fragments, which we endeavored to reduce by daily traction. Union was too firm for much improvement, and though he recovered with very good function, a permanent bowing of his arm remained (Fig. 63).

CASE II.—A little girl received a fracture of both bones of the forearm. At the first dressing, in the receiving ward, she was dressed in mid-pronation, but on her arrival in the dispensary, we immediately dressed her forearm in full supination which was continued until the time of her discharge.



FIG. 63.—Fracture of both bones of right forearm; union with deformity from sagging at point of fracture. Case I.

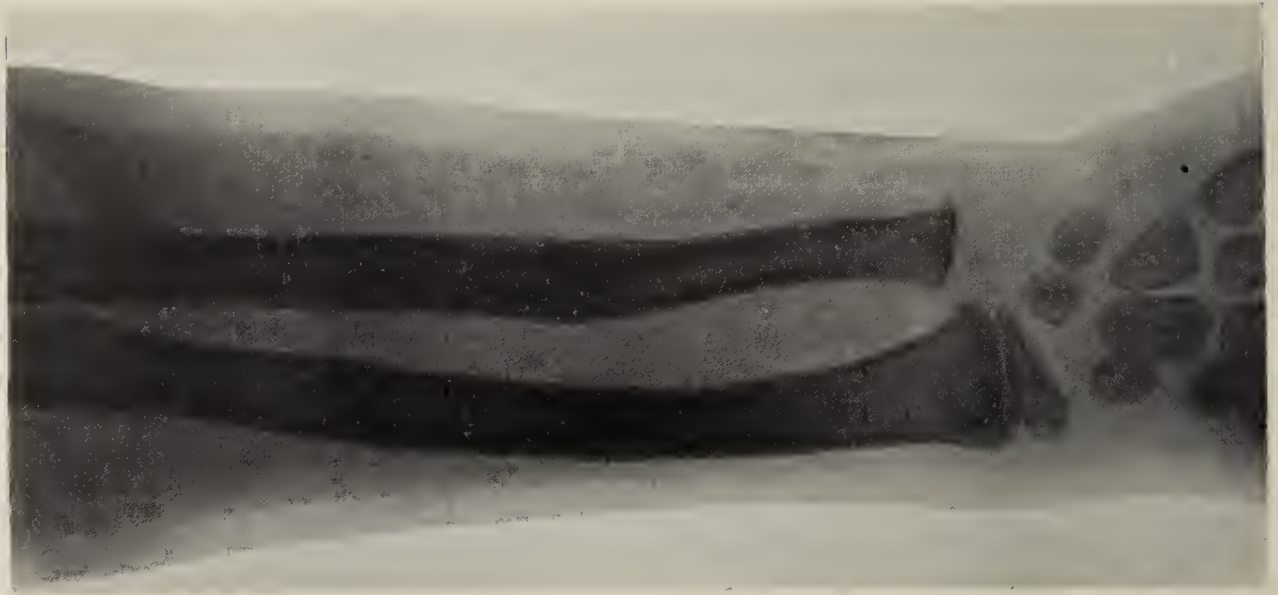


FIG. 64 *a*.—Fracture of both bones of forearm, which was dressed in full supination. Anteroposterior view. Case II.

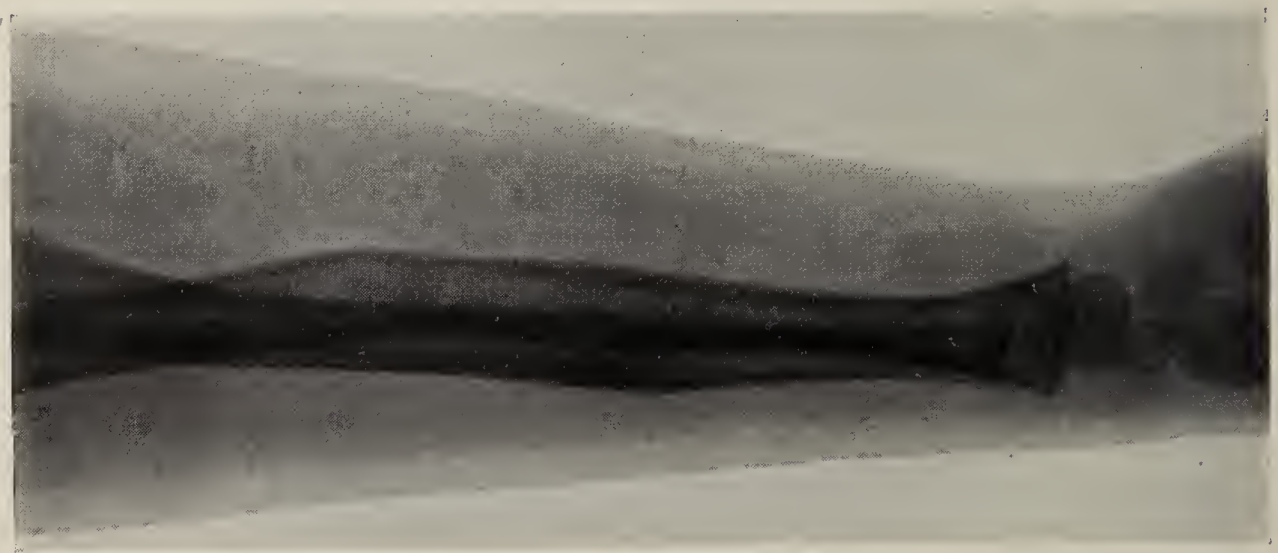


FIG. 64 *b*.—Fracture of both bones of forearm, which was dressed in full supination. Lateral view. Case II.

Her result was perfect in regard to function, and her forearm had absolutely no deformity. Figs. 64 *a* and *b* are from skiagraphs made eight months after injury.

In concluding, one may draw from these cases, which are typical examples of many others observed, the facts that full supination does not interfere with function by encroaching on the interosseous space, and that this method of treatment tends to prevent deformity.

FRACTURE OF THE RADIUS ABOVE THE ATTACHMENT OF THE PRONATOR QUADRATUS MUSCLE¹

BY EMORY G. ALEXANDER, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

IN reviewing the writings of the old surgeons, one is filled with admiration for their great work in the treatment of fractures. Their knowledge of the causes, deformities, action of muscles, manner of reduction and keeping reduced was truly remarkable. If these men could have had that valuable aid, the *x*-ray, in studying and treating their fractures, I am sure they would have handed down to the surgeons of today methods far in advance of those now in vogue.

In presenting this paper with accompanying *x*-ray plates, I hope to show that good approximation can often be obtained, if one will but persist and not be too hasty to resort to operative measures.

The following case is a fairly typical one of a fracture in this region. The patient was admitted to the Episcopal Hospital, to the service of Dr. H. C. Deaver, to whom I am indebted for the privilege of treating and reporting the case.

CASE I.—W. D., male, aged forty-one years. Ten days before admission, while at work, patient fell eight feet and as the result of direct force sustained a comminuted fracture of the radius above the attachment of the pronator quadratus muscle.

¹ Read before the Philadelphia Academy of Surgery, February 5, 1912. Reprinted from *Annals of Surgery* for June, 1912.

The attending physician, failing to get good approximation of the fragments, referred the case to the Episcopal Hospital for an operation.

The first *x*-ray showed the upper fragment abducted and rotated outward and the lower fragment strongly pulled over toward the ulna.

The patient had been under the care of a very skilful physician, who had first treated the fracture in the usual semipronated position with long palmar and short dorsal splints, and later on a Bond splint, without gaining a good approximation of the fragments.

We had made an internal angular splint (Fig. 65), somewhat pistol-shaped at the wrist, so as to strongly adduct the hand, hoping through the action of the external lateral ligament, and possibly the cartilage and the carpal bones, to pull or force the lower fragment in position.

The *x*-ray of the bones in this position showed that our efforts had not been entirely successful (Fig. 66). The failure seemed to be due to the upper fragment, as this was not affected by the position or adduction of the hand. As the lower fragment seemed to occupy almost a normal position, our efforts at reduction were next directed to the upper fragment.

To overcome its deformity, the arm was placed on an anterior angular splint, likewise pistol-shaped at the wrist (Fig. 67), and as in the previous dressing a short straight splint was applied posteriorly from the elbow to the wrist (Fig. 68). This changed the arm from a semipronated to a supinated position and relaxed the flexors of the forearm, thus producing one of the fundamental principles in the treatment of any fracture, muscular relaxation. It also supinated the lower fragment of the radius, bringing it in apposition to the upper fragment. The *x*-ray of the fracture in this position showed the bones to be in perfect alignment (Fig. 69). Fig. 70 shows end result.

As some difficulty had been encountered in gaining this approximation and as the fracture had been frequently disturbed during the ten days prior to admission to the hospital, and as there was no attempt at union, the splints were left on for twelve days. During these twelve days the bandages were frequently removed, without disturbing the splints, which were held in place by adhesive plaster, the soft parts inspected and bathed with alcohol and gentle passive motion given the fingers.

I believe this was unnecessarily long to keep the hand adducted as a few days in this position would have been sufficient. At the

expiration of the twelve days a short straight posterior splint combined with an anterior angular splint, straight at the wrist, was applied. This latter splint should be substituted as soon as possible, as it places the hand in a more comfortable position and minimizes the chances of a stiff wrist.

The method sometimes used of treating fractures of the forearm in a semi-pronated position with a small interosseous padded splint to force the fragments apart is a dangerous one, as so much force is required that pressure ulceration is apt to occur.

Caswell, in referring to this manner of treatment, says: "If useful, intolerable; if tolerable, useless."¹

In a previous case (Fig. 71) of fracture of the radius above the pronator quadratus, an interosseous padded splint was used correcting to a slight degree the deformity (Fig. 73). This treatment was discontinued, as the interosseous splint produced superficial ulceration. Later the arm was placed on an internal angular splint, pistol-shaped at the wrist, to strongly adduct the hand (Fig. 72). This position produced almost a perfect approximation, except for the upper fragment, which was tilted forward by the action of the biceps.

In treating this fracture, not only should one try to get perfect alignment, but also, as Lonsdale urged, "keep the fragments in their normal position as to their axis."²

Nélaton, in speaking of fractures of the lower end of the radius, says that Dupuytren laid great stress on the importance of overcoming radial displacement of the lower fragment. He used palmar and dorsal splints, as for fracture of both bones, and after they were applied added along the ulnar border of the forearm and hand an iron band, bent on the flat at the wrist, so as to draw the hand strongly to the ulnar side by means of tension on the external lateral ligament of the wrist.³

Amesbury, in speaking of fractures of the base of the radius, says not to allow the splint along the ulnar border to

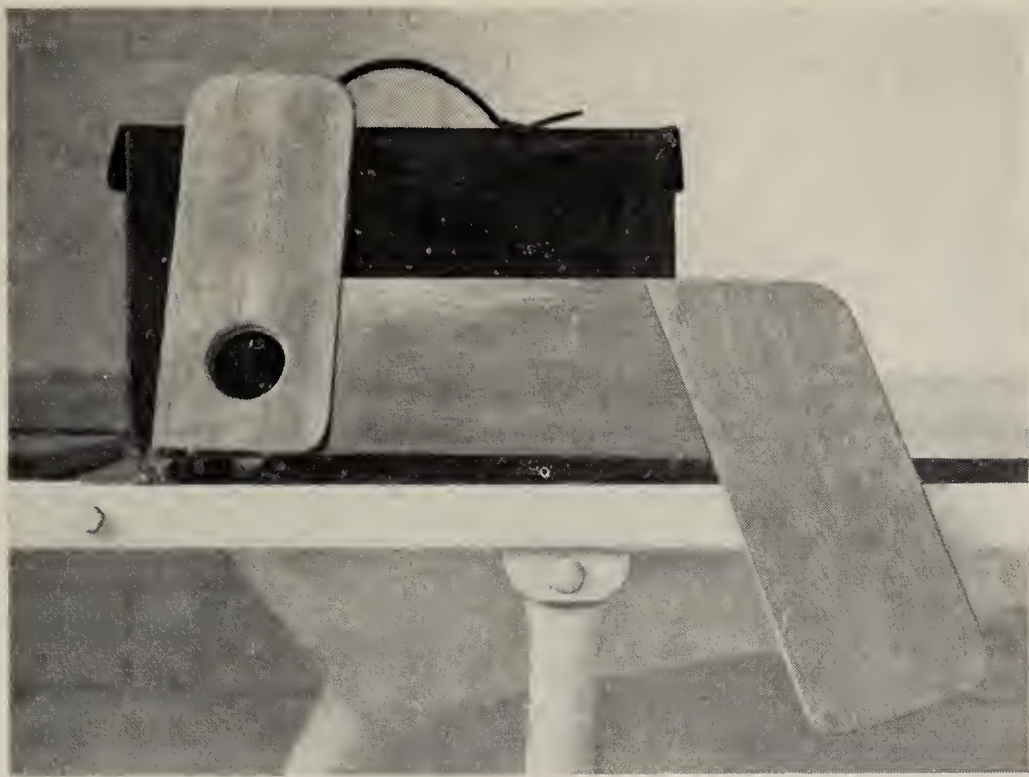


FIG. 65.—Internal angular splint to secure adduction of hand.



FIG. 66.—Arm on internal angular splint, pistol shaped at wrist; hand strongly adducted; deformity much improved.

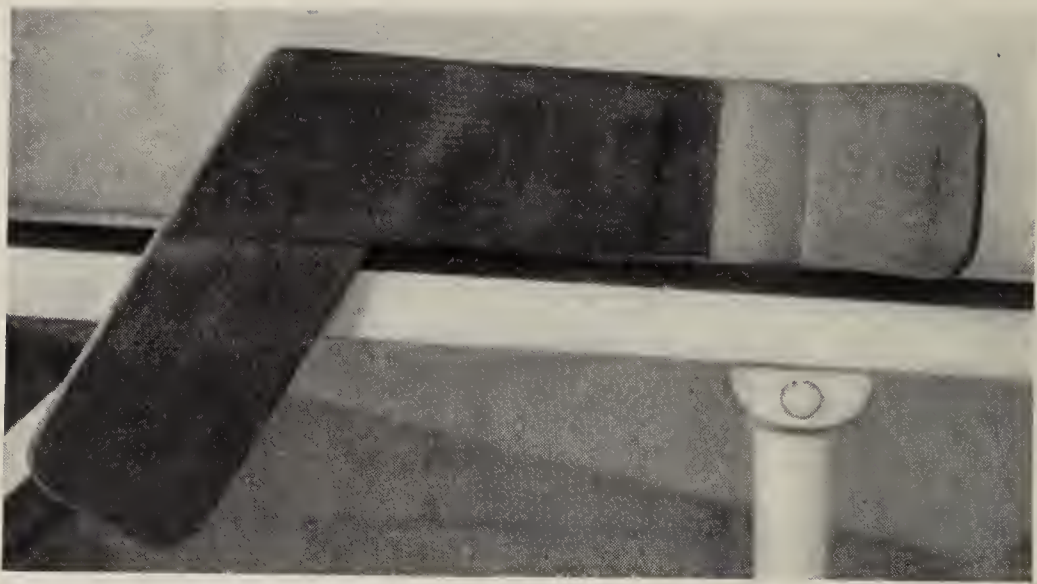


FIG. 67.—Anterior angular splint to procure adduction of hand.



FIG. 68.—Position of arm as shown in x-ray plate (Fig. 69).



FIG. 69.—Arm on anterior angular splint, pistol shaped at wrist; hand adducted; perfect alignment.



FIG. 70.—End result of Fig. 66.

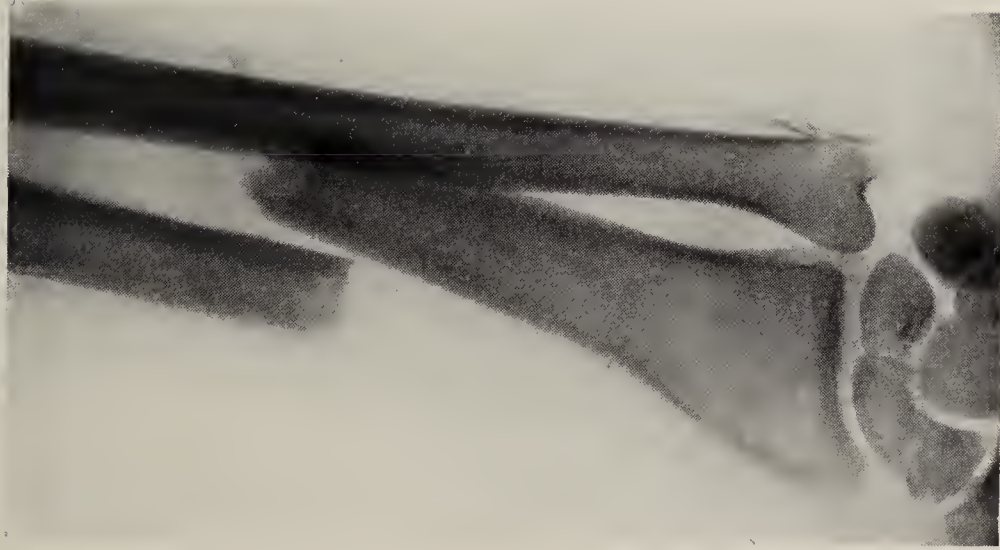


FIG. 71.—Fracture of radius with typical deformity.

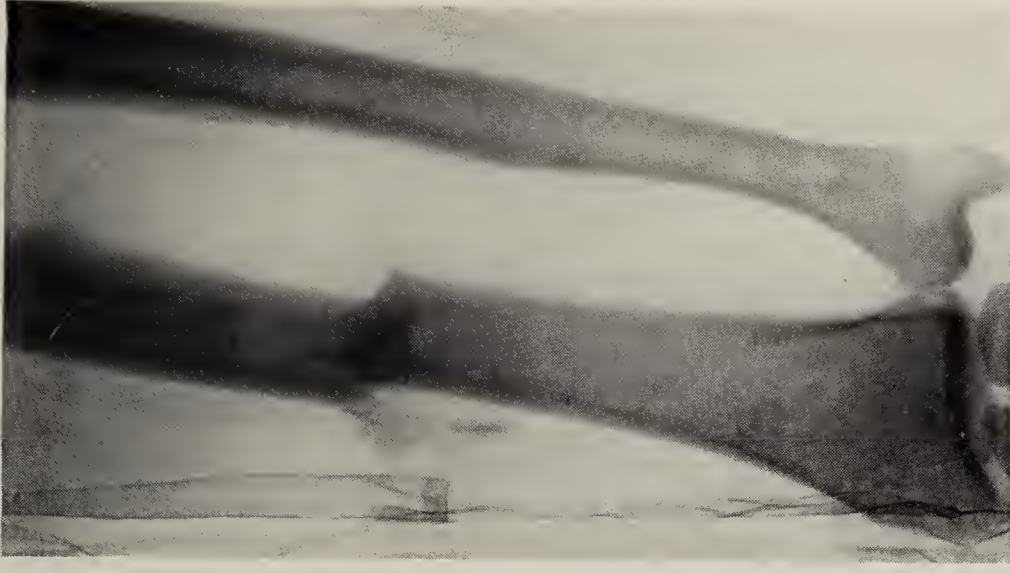


FIG. 72.—Arm semipronated; hand adducted; upper fragment tilted forward by action of biceps.



FIG. 73.—Deformity slightly corrected by interosseous padded splint; intolerable to patient; produced pressure ulceration.

extend lower than the wrist. He says: "The hand should be suffered to drop as low as possible before it is confined to the flat part of the back of the splint." "If the hand be confined down in this way it will act as a lever upon the carpal portion of the radius and tend to raise it."⁴

Lonsdale, after describing Dupuytren's splint to secure adduction of the hand in fractures of the lower part of the shaft of the radius, says: "When the position of extreme supination is employed, neither the radial nor the ulnar splint is necessary, for then the portions of bone have not the same disposition to fall toward the ulna."⁵

According to Packard, Nélaton's dorsal splint for securing adduction of the hand was originally described by Goyrand in 1836.⁶

I wish to thank the radiographer of the Episcopal Hospital, Dr. T. S. Stewart and his assistant, Dr. A. R. Wilkinson, for the many excellent x-rays of these fractures.

REFERENCES

1. Caswell. Holmes' System of Surgery, Am. ed., i, 861, as quoted by Packard.
2. Packard. Ashhurst's International Encyclopedia of Surgery, iv, 164.
3. Nélaton. Pathologie Chirurgicale, Paris, 1844, i, 745.
4. Amesbury. Practical Remarks on the Nature and Treatment of Fractures, London, 1831, ii, 604.
5. Lonsdale. Practical Treatise on Fractures, London, 1838, p. 148.
6. Packard. Ashhurst's International Encyclopedia of Surgery, 1884, vol. iv.

FRACTURE OF THE PATELLA, WITH A REPORT OF FIFTY-SIX CASES¹

BY EMORY G. ALEXANDER, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

THIS paper comprises a study of 56 cases of fracture of the patella, admitted to the Episcopal Hospital since the year 1905.

To Drs. Neilson, Deaver, Davis, and Frazier, to whose services these patients were admitted, I am indebted for the privilege of reporting these cases. To Dr. H. C. Deaver I am especially indebted for the privilege of operating upon several of these cases, and to his wise counsel, especially in suggestions of operative technique and after-treatment, do I owe much of the success gained.

The primary object of this paper is to discuss the operative technique and after-treatment, therefore little shall be said of the causes, varieties, diagnosis, and symptoms of this very important and interesting fracture.

Of the 56 cases tabulated, 37 were in males, while 19 occurred in females, a ratio of almost two to one. There is no anatomical explanation for the more frequent occurrence of this fracture among males, and, in all probability, it is due to their greater exposure and activity.

The ages in this series range from 18, the youngest, to 77, the oldest. I have arbitrarily classified them according to age as follows:

¹ Read before the Philadelphia Academy of Surgery, December 5, 1910. Reprinted from *Annals of Surgery*, April, 1911.

Four occurred between the ages of ten and twenty; 11 between twenty-one and thirty; 19 between thirty-one and forty; 14 between forty-one and fifty; 6 between fifty-one and sixty; 1 between sixty-one and seventy, and 1 between seventy-one and eighty.

The greatest frequency occurred between the ages of thirty-one and forty years; the period, certainly, of the greatest business activity. It is rather unusual for this fracture to occur under twenty years of age. This is probably due to the bony condition or better muscular control of the young, they being less apt to slip or fall. The youngest patient that I have any personal knowledge of was a boy aged twelve years. This case was operated upon with excellent result by Dr. H. C. Deaver, at the Children's Hospital of the Mary J. Drexel Home.

Of the 37 males, 19 broke their left patella, while 18 broke the right. Of the females, 11 broke the left, and 8 the right patella.

It appears from this, although the numbers are almost equal, the ratio being larger in the females, that the left patella is more apt to be fractured. Anatomists claim that no asymmetry exists in the lower extremity. Is this fracture purely an accident or is there some cause for its more frequent occurrence in the left patella? Some greater muscular development, a longer step with one leg, or a firmer tread with one foot should be thought of.

The causes given in this series were: "fell," 27; "slipped," 27; "kicked," 2. The histories were somewhat indefinite on this point and I could find only 9 that actually fell from a distance, so whether the "fell" meant a slip and a fall, or a fall from a distance, I cannot say. I am inclined, however, to think the former the case. Several of the patients stated that they slipped, heard something break, and fell.

A great majority of these fractures were of the usual transverse variety, with the large fragment above. A few of this transverse variety showed the reverse to be true,

the large fragment being below. Forty-nine cases were of this combined transverse type. Six cases were comminuted; these occurred in two that were kicked, and in four that fell from a distance. The number of fragments in this latter type varied from three to many. No compound fracture occurred in this series.

I have taken a picture of a normal patella in three positions: with leg extended, semiflexed, and acutely flexed. The patella with the leg semiflexed is seen at the highest point of the condyle of the femur, and it is with the leg in this position that the fracture usually occurs. A sudden strain on the leg, with a violent contracture of the quadriceps extensor muscle, snaps the patella at its weakest and most unsupported point, the lower third.

The symptoms and diagnosis I shall omit, as there is nothing I can add to that already known of the former, and the latter is usually quite easy.

Little shall be said of the non-operative treatment, as all agree that by this method nothing but fibrous union can be hoped for, with more or less separation of the fragments. Certainly in the great majority of instances the results are far inferior to those of the open method of treatment. There are, of course, certain cases that must be treated by this method, as the aged, those in whom some constitutional condition contraindicates operation, or those who, although good operative risks, refuse to be operated upon. Comminuted fractures, with no separation or tilting of the fragments, and, in all probability, no tear in the fibrous expansion of the quadriceps tendon, fascia lata, and joint capsule, may also be treated by the conservative non-operative method. Of the many conservative methods of treating this fracture, that employed by the late Dr. Agnew is probably as good as any.

I am fully aware that many think the open method of treatment a dangerous one and one that should be done only by a skilled operator. They are willing to operate themselves,

but unwilling to teach it. It is dangerous for an occasional operator and one unfamiliar with asepsis and operative technique to do any major operation. I believe that the open method should be taught, but, at the same time, the physician and occasional operator should be alive to the fact that an operation for fracture of the patella is not a minor one, involving, as it does, the largest and at the same time one of the weakest joints in the body, and that if infection does occur, it may end most disastrously. Consequently it is an operation accompanied with a certain definite risk, and one probably attended by much more danger than a simple appendectomy, as the synovial membrane does not seem to possess the protective power of the peritoneum in taking care of a slight infection.

The best time to operate is now thought to be after all oozing has ceased, after the exudate has reached its height and has even begun to subside, and the tissues have had time to become sealed off. This process usually takes from six to ten days, but it can be hastened somewhat by placing the leg on a posterior splint, with elevation, and the application of an ice bag. In reviewing the histories of these cases, the temperature charts, as a whole, failed to show any marked difference between those operated upon early and those in whom the operation was delayed. The immediate success of these cases depends entirely on whether or not infection occurs. This, I believe, rests between the resistance of the tissues, the virulence of the infection, if one does occur, the preparation of the patient, and the operative technique. Dr. Murphy has pointed out that in an early operation the surgeon is working in tissues somewhat devitalized and, therefore, less resisting and more apt to become infected.

In a letter, which I quote with his permission, Dr. Murphy says: "My reason for postponing the operation for six to ten days is to give the synovial membrane an opportunity to react to the irritation of the trauma and the irritation of the blood-clot in the joint. This reaction produces a coffer-

damming of the lymph spaces in the subendothelial layer of the synovial membrane, and lessens the danger of infection very materially."

"We resorted to an injection of 10 c.c. of formalin and glycerine into the joint, immediately after the fracture. This produces a chemical irritation, increases the number of polymorphonuclear leukocytes in the joint, and increases the constitutional polymorphonuclear reaction. It also cofferdams the lymph spaces and insures a prophylaxis against infection. The operation is then done five to seven days after this injection."

Theoretically, with perfect technique, there is nothing to gain by delay other than to allow the oozing, especially from the torn synovial membrane, to cease, as this in an early operation can be quite annoying. Practically, however, we know that our technique is not always perfect and that infection does sometimes occur. This is lessened by a late operation and, likewise, I believe, the patient suffers much less the first few days after the operation, and the convalescence is shortened, as there is less local reaction.

The incision should be elliptical. It makes little difference if the convexity is above or below. Some surgeons claim the convexity should be above, as it takes the scar away from the knee and is less apt to cause pain with the patient in the kneeling posture. Of the cases I was able to follow, I was unable to substantiate this claim. I think the incision should be an elliptical one, preferably below, as a greater exposure can be obtained well away from the line of fracture, and it lessens the chance of infection and after-complications, especially if silver wire is used. The incision should be carried well down on either side, as I shall point out later, to permit drainage. The next step in the operation, after reflecting well the skin flap, is to divide the prepatellar bursa and fascia lata. The clots are now swabbed out with dry gauze, no fluid being used, and if any irrigation is necessary, only saline solution or sterile water should be employed, as bi-

chloride or other devitalizing or irritating agents are apt to increase the flow of serum and favor infection. The reflected tendon over the broken edges is now retracted, the frayed ends rounded, and by blunt dissection separated a short distance from the margin of the fracture. Often this is impossible on account of the small fragment. The raw surface of the patella should next be freshened, especially in late operations, to get rid of the adherent organizing clot. With a hand drill, beginning in the centre of the upper fragment about one centimetre from the margin of the fracture, a hole is drilled obliquely downward so as to emerge on the broken surface just at the point where the dense cancellous tissue and thin compact lamina unite. A similar opening is drilled in the lower fragment. Through these two holes a silver wire is passed, the fragments are brought together as accurately as possible, and the wires twisted one or two times; the redundant wire is cut off, and the twisted ends that remain are reflected upward under the tendon and gently hammered down. Some operators use two wire sutures, one being placed on either side of the patella. The tendon is next sutured with chromic gut, and likewise the torn fibrous expansion of the quadriceps tendon, fascia lata, and joint capsule. At the lower angle of the wound, if the rent in the fibrous expansion of the quadriceps tendon, fascia lata, and joint capsule does not extend so far down, an opening should be made to permit drainage. The prepatellar bursa and fascia lata are next sutured with chromic gut and the skin by interrupted silkworm gut sutures. The skin wound should not be sewed too tight, neither should too many stitches be applied. The angles of the wound corresponding to the openings in the fibrous expansion of the quadriceps tendon, fascia lata, and joint capsule should be left open. As a rule, this procures sufficient drainage, but if there has been much oozing, a few strands of silkworm gut or a small piece of rubber tissue can be inserted. The leg is now placed on a well-padded, slightly convex, posterior splint until the patient has fully recovered

from the effects of the anæsthetic, when the splint is removed and the leg is placed on a pillow.

The point that I wish to emphasize in the operative technique is the advantage of silver wire. Out of the thirty cases that I have been able to follow, four refractured the patella; three of these were sutured with absorbable material. The one that refractured with wire was due to a fall downstairs, and so great was the strain thrown upon the patella, that the wire cut through the upper fragment. Silver wire is certainly the most aseptic and at the same time the strongest suture material that can be used. The only disadvantage is that it occasionally has to be removed. Removal was necessary in three cases in this series. This undoubtedly can be avoided, provided no infection occurs, but even if skin infection—the most common in this operation—does occur, if the skin incision is well away from the line of fracture, and if the ends of the wire are not left too long and are well covered by the tendon, fascia lata, and prepatellar bursa, removal of the wire may not be necessary. If the wire has to be removed, however, it can be easily done with little inconvenience to the patient. That wire causes softening of the bone around the opening is highly improbable unless some infection occurs. Wire is certainly no more irritating than any other material used in these cases, as kangaroo tendon, chromic gut, or even, as has been used, silkworm gut.

Cotton, in his excellent book on "Dislocations and Joint Fractures," states that refracture after the eighth week is rare. Only one of this series occurred in that time; the others ranging from four months to four years.

Is bony union obtained in a fracture of the patella? Some surgeons claim not. Personally, I have never examined a sutured fractured patella under the microscope and cannot say. In one of these cases I removed a wire one year after operation. I took the opportunity, clinically, to examine the union and to all appearances it was bony. In this case,

even though there had been a slight skin infection followed by a persistent sinus for several months, the bone did not appear soft around the wire, and it took quite a "tug" to dislodge it. As shown by the refractures occurring in this series, the bony union, if one is obtained, is not strong. Why then not reinforce this with a non-absorbable suture?

The after-treatment of these cases is most important. The splint is removed as soon as the patient has recovered from the effects of the anæsthetic, or, preferably, it can be left on through the first restless night following the operation. On its removal, the leg, slightly flexed, is placed on a pillow. Gentle passive motion is begun in a day or so. As it is possible to move the leg through an angle of five or ten degrees without moving the patella, this much motion is taken advantage of. The passive motion is gradually increased so that by the third week the leg can be flexed to a right angle. In the last case that I operated upon, the patient could flex the leg to a right angle on the tenth day, was allowed out of bed on the twelfth, and walked the next day. He was discharged, walking, from the hospital on the sixteenth day. It is a mistake to keep the leg for weeks on a splint or in a cast. Not only does the patient lose much time by the delay, but the muscles become atrophied and the knee more or less ankylosed. These patients will tell you that they were a year getting a useful limb. Of the ultimate end results, say one year after operation, there is little to choose between. All that I was able to follow got a fairly good functioning result. Some complained of a little stiffness or weakness in the knee on flexion or extension. A few complained of pain in the knee before a storm. On the whole, all showed excellent results; especially was this true of the five private cases operated upon by Dr. Deaver. These patients were treated by the above method, and so excellent were the results, that one would never know, except on close examination, that they had a fracture of the patella. The others, in all prob-

ability, would have had as good a result if they could have been properly carried through the late after-treatment.

In this series, two cases died, both from sepsis. One was operated upon on the fourth day, the other on the thirty-fifth day following the accident. The first, a woman, had an abortion ten days before the accident, and when operated upon, unknown to the surgeon, had a bad discharge from her uterus. Whether this patient died from a primary infection or one occurring through her blood, it is hard to say. Her knee did not show much inflammatory change for several days after the operation, although she was profoundly septic. Repeated blood cultures were negative. A culture taken from the knee, however, showed a bacillus morphologically resembling the Klebs-Löffler. Everything possible was done to save this patient's life. Her leg was amputated three and one-half months after the primary operation, but she died two weeks later of exhaustion. The other death occurred in a man who was operated upon five weeks after the accident. I saw the operation performed and the technique was apparently faultless, but evidently some error occurred, for the knee became infected, and the patient died two months later of sepsis.

The majority of cases operated upon showed a febrile reaction ranging from 99° to 102° a day or so after the operation, but the fever usually subsided by the fourth to the sixth day.

In those cases badly infected following the operation, the best chance of saving the patient's life is by early laying open the joint and packing with iodoform gauze.

Some surgeons claim that in comminuted fractures a conservative method of treatment should be used. This rule, as all others, has its exceptions. In one fracture that I operated upon, due to a kick, the patient fell after the blow and evidently tore the fibrous expansion of the quadriceps tendon. In another case, due to a fall from a distance, there was little

tear of the fibrous expansion of the quadriceps tendon, but the fragments were tilted and separated by the effusion and clot. These two classes of cases should certainly be operated upon; in the first instance to repair the torn fibrous expansion of the quadriceps tendon, fascia lata, and joint capsule, and in the second, to adjust the fragments and to turn out the clot. In comminuted fractures, especially if broken in many pieces, suturing the bone is often impossible, and the best that can be done is to suture the tendon and carry the patient through a prolonged convalescence.

In the letter referred to above, Dr. Murphy also says: "In cases where the patella is badly fragmented, we believe the use of a flap three-fourths of an inch wide and four and one-half inches long, from the central portion of the quadriceps tendon, passed over the patella and inserted into the ligamentum patellæ by splitting it and looping it half way around, is the most secure means of holding the patella. It does not then involve the traumas in the joint nor the presence of foreign material, such as wire or plates. It is one of the simplest means of treating these fractures, and I believe one of the most secure, following out the plan I do in my cases of resection of the patella for tuberculosis."

In fracture of the patella, if bony union does occur, close approximation of the fragments is essential. This close approximation cannot always be gained by simply suturing the tendon and not the patella, as effusion or movement may dislodge the fragments. In suturing with an absorbable material, a close approximation is possible, but often these sutures soften, elongate, become untied or even break; especially is this so, if close approximation is not obtained and the fragments move independently of each other. In using absorbable material, passive motion must be delayed and the patient is compelled to pass through a slow convalescence, followed by more or less stiffness of the joint that usually lasts for several months, to say nothing of the loss of time which many of them can ill afford.

Total number of fractures of the patella	56
Males	37
Females	19
Males fracturing right patella	18
Males fracturing left patella	19
Females fracturing right patella	8
Females fracturing left patella	11
Variety: transverse fracture	50
Comminuted fracture	6
Suture: silkworm gut	1
Chromic gut	15
Kangaroo tendon	2
Silver wire	31
After-treatment: plaster case	15
Splint	8
Splint and case	9
Pillow	17
Splint (not operated)	7
Causes: slipped	27
Fell	27
Kicked	2
Tendon alone sutured	3
Refractures: absorbable suture	3
Wire suture	1
Operated upon	49
Not operated upon	7

REPORT OF CASES

E. W., aged forty-two years; female. Transverse fracture of patella of left knee; caused by slip. Operation six days later; silkworm gut suture; cast. In hospital fifty-seven days. Highest temperature 99.3. Recovered.

S. B., aged twenty-years; male. Transverse fracture of patella of right knee; caused by fall. Operation one day later; chromic gut suture; cast. In hospital twenty-seven days. Highest temperature 100.3. Recovered.

S. B., aged twenty-seven years; female. Transverse fracture of patella of right knee; caused by slip. Operation two days later; wire suture; splint and cast. In hospital forty-five days. Highest temperature 99.4. Recovered.

P. N., aged forty-three years; male. Comminuted fracture of patella of right knee; caused by fall. Not operated; splint. In hospital fifty days. Recovered.

J. S., aged forty-eight years; male. Transverse fracture of patella of left knee; caused by slip. Operation two days later; chromic gut suture; splint and cast. In hospital thirty-three days. Highest temperature 101.2. Recovered.

A. F., aged thirty-eight years; male. Transverse fracture of patella of left knee; caused by slip. Operation two days later; chromic gut suture; cast. In hospital eighty-five days. Highest temperature 100.1. Recovered.

A. S., aged fifty-five years; female. Transverse fracture of patella of right knee; caused by slip. Operation twenty days later; wire suture; splint. In hospital fifty days. Highest temperature 100.1. Recovered.

A. N., aged sixty-seven years; female. Comminuted fracture of patella of right knee; caused by slip. Not operated; splint. In hospital thirty-five days. Recovered.

J. B., aged twenty-seven years; male. Transverse fracture of patella of left knee; caused by slip. Operation two days later; kangaroo tendon suture; splint and cast. In hospital fourteen days. Highest temperature 100. Recovered.

H. W., aged forty years; male. Comminuted fracture of patella of right knee; caused by kick. Not operated; splint. In hospital thirty days. Recovered.

H. B., aged thirty-eight years; female. Transverse fracture of patella of left knee; caused by fall. Operation three days later; chromic gut suture; cast. In hospital twenty-five days. Highest temperature 100.1. Recovered.

W. W., aged forty-four years; male. Comminuted fracture of patella of left knee; caused by fall. Operation one day later; chromic gut suture; cast. In hospital thirty-one days. Highest temperature 100. Recovered.

G. O., aged thirty-seven years; male. Transverse fracture of patella of left knee; caused by slip. Operation fourteen days later; chromic gut suture; splint. In hospital thirty-five days. Highest temperature 102. Recovered.

A. S., aged fifty-four years; female. Transverse fracture of patella of left knee; caused by fall. Not operated; splint.

H. B., aged thirty-eight years; female. Transverse fracture of patella of left knee; caused by fall. Operation four days later; wire suture; pillow. In hospital twenty-eight days. Highest temperature 100.1. Recovered.

H. R., aged thirty years; male. Transverse fracture of patella of left knee; caused by slip. Not operated; splint.

W. W., aged forty-four years; male. Transverse fracture of patella of left knee; caused by fall. Operation four days later; wire suture; pillow. In hospital thirty-two days. Highest temperature 99.1. Recovered.

J. B., aged twenty-seven years; male. Transverse fracture of patella of left knee; caused by fall. Operation three days later; kangaroo tendon suture; cast. In hospital thirty-four days. Highest temperature 100.2. Recovered.

F. S., aged twenty-eight years; female. Transverse fracture of patella of left knee; caused by fall. Operation four days later; wire suture; pillow. In hospital twenty-five days. Highest temperature 100. Recovered.

R. G., aged thirty-two years; male. Transverse fracture of patella of right knee; caused by fall. Operation one day later; wire suture; splint. In hospital thirty-five days. Highest temperature 100.1. Recovered.

M. H., aged seventy-seven years; female. Transverse fracture of patella of left knee; caused by fall. Not operated; splint.

E. G., aged fifty years; male. Transverse fracture of patella of right knee; caused by fall. Operation four days later; chromic gut suture; cast. In hospital twenty-six days. Highest temperature 100.2. Recovered.

F. H., aged thirty-six years; male. Transverse fracture of patella of left knee; caused by slip. Not operated; splint.

L. B., aged sixty years; male. Transverse fracture of patella of right knee; caused by fall. Operation eighteen days later; wire suture; pillow. In hospital forty-five days. Highest temperature 100.1. Recovered.

W. K., aged forty-four years; male. Transvers fracture of patella of left knee; caused by slip. Operation four days later; wire suture; splint. In hospital thirty-five days. Highest temperature 100.2. Recovered.

E. S., aged twenty-seven years; female. Transverse fracture of patella of left knee; caused by slip. Operation four days later; wire suture; pillow. In hospital sixteen days. Highest temperature 99.4. Recovered.

W. H., aged forty years; male. Comminuted fracture of patella of right knee; caused by fall. Operation two days later; wire suture; splint and cast. In hospital twenty-nine days. Highest temperature 100.1. Recovered.

E. S., aged forty-five years; female. Transverse fracture of patella of right knee; caused by fall. Operation one day later;

wire suture; splint and cast. In hospital forty-six days. Highest temperature 100. Recovered.

J. S., aged thirty-seven years; male. Transverse fracture of patella of left knee; caused by fall. Operation four days later; wire suture; cast. In hospital twenty-five days. Highest temperature 100.2. Recovered.

C. D., aged thirty-one years; female. Transverse fracture of patella of left knee; caused by slip. Operation two days later; chromic gut suture; cast. In hospital forty-one days. Highest temperature 100.1. Recovered.

W. M., aged forty years; male. Transverse fracture of patella of left knee; caused by slip. Operation one day later; wire suture; cast. In hospital fifteen days. Highest temperature 99.4. Recovered.

N. R., aged thirty-five years; male. Transverse fracture of patella of right knee; caused by fall. Operation one day later; wire suture; pillow. In hospital seventeen days. Highest temperature 100.1. Recovered.

N. R., aged thirty-five years; male. Transverse fracture of patella of right knee; caused by fall. Operation two days later; wire suture; pillow. In hospital twelve days. Highest temperature 99.3. Recovered.

E. W., aged thirty-four years; female. Transverse fracture of patella of right knee; caused by slip. Operation one day later; wire suture; cast. In hospital thirty-three days. Highest temperature 101. Recovered.

J. F., aged forty-two years; male. Transverse fracture of patella of right knee; caused by fall. Operation three days later; chromic gut suture; splint and cast. In hospital thirty-two days. Highest temperature 99.4. Recovered.

C. M., aged thirty years; male. Transverse fracture of patella of right knee; caused by slip. Operation six days later; chromic gut suture; splint. In hospital forty-one days. Highest temperature 101. Recovered.

W. L., aged eighteen years; male. Transverse fracture of patella of right knee; caused by slip. Operation seventeen days later; wire suture; pillow. In hospital twenty-five days. Highest temperature 99.3. Recovered.

E. K., aged fifty-five years; male. Transverse fracture of patella of right knee; caused by slip. Operation thirty-five days later; wire suture; splint. In hospital sixty-seven days. Highest temperature 105. Died.

J. F., aged fifty-three years; male. Transverse fracture of patella of left knee; caused by fall. Operation seven days later; wire suture; pillow. In hospital twenty-seven days. Highest temperature 99.4. Recovered.

J. W., aged fifty-five years; male. Transverse fracture of patella of right knee; caused by slip. Operation twelve days later; wire suture; pillow. In hospital thirty-five days. Highest temperature 100.1. Recovered.

F. T., aged thirty-five years; female. Transverse fracture of patella of right knee; caused by fall. Operation four days later; wire suture; pillow. In hospital one hundred and fifteen days. Highest temperature 105.3. Died.

J. R., aged nineteen years; male. Comminuted fracture of patella of left knee; caused by kick. Operation four days later; wire suture; pillow. In hospital thirty-six days. Highest temperature 100.4. Recovered.

A. T., aged forty-five years; male. Transverse fracture of patella of right knee; caused by slip. Operation five days later; wire suture; splint and cast. In hospital twenty-eight days. Highest temperature 100.3. Recovered.

A. D., aged forty years; female. Transverse fracture of patella of right knee; caused by slip. Operation five days later; chromic gut suture; splint. In hospital forty-five days. Highest temperature 100.2. Recovered.

J. O., aged thirty-three years; male. Transverse fracture of patella of right knee; caused by fall. Operation two days later; chromic gut suture; cast. In hospital twenty-two days. Highest temperature 99.4. Recovered.

F. M., aged thirty years; female. Transverse fracture of patella of left knee; caused by slip. Operation four days later; wire suture; pillow. In hospital forty-four days. Highest temperature 100.3. Recovered.

E. W., aged forty-nine years; male. Transverse fracture of patella of right knee; caused by slip. Operation eight days later; wire suture; pillow. In hospital thirty days. Highest temperature 100.4. Recovered.

J. S., aged thirty-seven years; male. Transverse fracture of patella of left knee; caused by fall. Operation six days later; chromic gut suture; splint. In hospital twenty-nine days. Highest temperature 100.1. Recovered.

T. G., aged twenty-two years; male. Transverse fracture of patella of left knee; caused by slip. Operation four days later;

wire suture; pillow. In hospital thirty days. Highest temperature 101.4. Recovered.

F. H., aged twenty years; female. Transverse fracture of patella of right knee; caused by fall. Operation nine days later; wire suture; cast. In hospital thirty-eight days. Highest temperature 100. Recovered.

L. W., aged twenty-eight years; female. Transverse fracture of patella of left knee; caused by slip. Operation one day later; wire suture; pillow. In hospital thirty-four days. Highest temperature 100.1. Recovered.

G. E., aged forty-one years; male. Transverse fracture of patella of left knee; caused by fall. Operation five days later; chromic gut suture; cast. In hospital forty-two days. Highest temperature 101. Recovered.

M. A., aged thirty-nine years; female. Transverse fracture of patella of left knee; caused by slip. Operation five days later; chromic gut suture; cast. In hospital sixty-one days. Highest temperature 100.4. Recovered.

D. M., aged forty-four years; male. Transverse fracture of patella of left knee; caused by slip. Operation eight days later; wire suture; pillow. In hospital twenty-five days. Highest temperature 99.4. Recovered.

W. M., aged twenty-eight years; male. Transverse fracture of patella of right knee; caused by fall. Operation two days later; wire suture; splint and cast. Highest temperature 101.3. Recovered.

G. H., aged forty-two years; male. Transverse fracture of patella of left knee; caused by slip. Operation nine days later; wire suture; splint and cast. Highest temperature 98.3. Recovered.

CASES OF ARTHROTOMY OF THE KNEE¹

By ASTLEY P. C. ASHHURST, M.D.
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1. For "*hydrops articuli*." *Perfect function eight years later.* James L., was a grave-digger, aged thirty years. He had never been ill, but one day in the summer of 1903, he had a sudden pain in his right knee, which continued all one day, and then left him never to return. In June, 1904, this knee began to feel clumsy and weak; he had not injured it, and it did not hurt him. After three months of increasing disability he came to the Orthopædic Hospital, service of Dr. G. G. Davis. Examination showed the knee irregularly swelled up by what was thought to be an intra-articular growth, of semifluid consistency; the patella was raised, and there was a marked prominence on each side of the tendon of the quadriceps extensor. The swelling felt "myxomatous" rather than fluid. On September 1, an aspirating needle was introduced, but no fluid could be withdrawn. On September 5, 1904, the man was etherized, and Dr. Ashhurst made an incision on each side of the patella; the joint contents were turbid serum; no flakes of lymph nor rice bodies were present. The under surface of the patella was eroded, as was the apposed surface of the femur; there was no roughening of the capsule. The joint was wiped out with iodoform gauze, and a large double drainage tube was passed from one side of the joint to the other. A sterile dressing and a posterior splint were applied.

The tubes were removed within a few days, as soon as all discharge ceased. On September 28, the incisions were healed, and the limb was put up in plaster of Paris. There had been no pain since operation. On October 13 the patient was discharged, still wearing the gypsum case. This was removed in November,

¹ Read before the Philadelphia Academy of Surgery, May 6, 1912. Reprinted from *Annals of Surgery*, October, 1912.

and an elastic bandage was worn for about a year. Some disability persisted for nearly a year.

In the summer of 1906, two years after operation, examination showed perfect function. He still wore an elastic knee-cap, and there was slight atrophy of the thigh.

At present, nearly eight years after operation, the patient is in perfect condition. Flexion and extension in the knee are normal; the patella is freely movable; there is no effusion; and the joint is symptomless. He is still digging graves, and never has had any pain or discomfort in the knee since the operation.

2. *For recently dislocated semilunar cartilage.* Florence S., aged twenty-four years, wore high-heeled shoes, and tripped and fell on the street. Her right knee was violently flexed, causing great pain; but with her hands she managed to straighten the leg out, and then found she could not bend it up again. She felt a tender lump on the outside of the knee, just below the femoral condyle. She was brought to the Episcopal Hospital the same day, August 29, 1911, and was admitted to the service of Dr. Chas. H. Frazier. Examination confirmed the above sad state of affairs. There was a tender lump below the external condyle, the knee was locked in extension, and could not be flexed. X-rays showed nothing. The next day Dr. Ashhurst operated. While the patient was being etherized, the lump, which up to that time had been immovable, suddenly disappeared, and motion in the joint became free. On opening the joint over the external semilunar cartilage, there was a gush of turbid synovial fluid. The external semilunar moved freely on the head of the tibia, but retained its attachments to the capsule, as well as anteriorly and posteriorly. No other lesions were observed. The loose cartilage was excised by the aid of a sharp hook and scissors, and the joint was closed without irrigation or drainage, and plaster-of-Paris dressing applied.

On the fourth day the temperature rose to 102° F., and there was a great deal of pain in the joint; but the pulse was only 85.

Three weeks after operation the gypsum case was removed, and the wound was found healed. Subsequently there was a little skin infection, but culture of the pus showed no growth, and smears showed no bacteria. One month after operation the knee could be flexed to 150 degrees. In November the patient, who had resumed her high-heeled shoes, fell again in the street, and suddenly flexed the injured knee to more than a right angle; this caused slight effusion, and she was laid up for a few days. But on subsidence of this, no disability remained, and she has now

recovered perfect function, and spends much of her time in dance-halls, as before her accident.

3. *For recurrent dislocation of semilunar cartilage.* John W., when aged twenty-three years, hurt his left knee playing football. It always troubled him since. In March, 1911, when aged thirty years, and twice between that date and September, 1911, he suffered from acute dislocation of the internal semilunar cartilage of this knee. Suddenly, following twist or sprain, a lump appears below the internal condyle, the knee locks in flexion, and when straightened out by force cannot be bent. Acute synovitis follows, and he is laid up for a few days. He was admitted to Dr. Frazier's service in the Episcopal Hospital three weeks after the last attack. On September 9, 1911, Dr. Ashhurst removed a frayed-out internal semilunar cartilage, which was entirely unattached except to the capsule and at its posterior end to the tibia. The whole cartilage was removed. The gypsum case was discarded three weeks after operation, and in two months the knee had become entirely normal and has remained so since, now eight months.

TREATMENT OF DISLOCATION OF THE HEAD OF THE RADIUS COMPLICATED BY FRACTURE OF THE ULNA¹

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IN small children so-called subluxation of the head of the radius ("pulled elbow") occurs not infrequently, usually from vertical traction on the forearm, as the child's caretaker helps or lifts him across an obstruction in the street. If the forearm is in supination this vertical traction tends to bring the forearm and arm into a straight line, causing momentary loss of the carrying angle; or forced pronation may pry the radius forward over the ulna as a fulcrum. Strong contraction of the biceps, on the child's part, no doubt aids materially in causing the displacement. The pathology of these slight injuries is not very definite; but the disability is quickly relieved by full supination of the forearm, followed by direct backward and inward pressure on the displaced head of the radius. The explanation of Duverney (1751), that the head of the radius is drawn downward and caught in the orbicular ligament, is accepted by Stimson as the most satisfactory of the numerous explanations which have been suggested; but at the risk of adding a new theory to many that are unsatisfactory, it has occurred to me that a very simple explanation may be that the head of the radius merely is "decentered," so to speak, on the capitellum of the humerus, and is held in this

¹ Read before the Philadelphia Academy of Surgery, May 6, 1912. Reprinted from *Annals of Surgery*, for October, 1912.

malposition by muscular spasm or ligamentous tension. Such an idea is expressed, but not very definitely, by Malgaigne. The deformity is scarcely appreciable, and the disability is so very easily corrected and has in some cases corrected itself with such facility, that it seems hard to accept a definite rupture of ligaments as a cause. Moreover, recurrence of the condition is not unusual, without recognizable trauma.

Of more interest are the complete luxations of the radial head, because in many cases they are irreducible. The head may indeed be forced back seemingly into place, but as soon as the dressings are discontinued, and full extension is allowed, it is seen that nothing has been gained, as re-luxation occurs. The explanation is that real reduction never was secured. If reduction had been secured, and the torn orbicular ligament had re-united in proper position in front of the neck of the bone, it does not seem likely that the action of the biceps could destroy Nature's efforts at repair with such amazing facility and the production of so little pain.

Anterior luxation of the head of the radius alone is considered by most writers a much rarer injury than the same lesion accompanied by fracture of the ulna. Perrin (*Paris Thesis*, 1909, p. 17) says that every year Kirmisson sees two or three instances of the latter injury; and he quotes with astonishment the statistics of Zieger (1901), who found only 15 out of 95 cases of anterior luxation, and only 16 out of 42 cases of outward luxation of the radial head were accompanied by fracture of the ulna. Under my own care, during the last ten years, I have had 23 cases of isolated fracture of the shaft of the ulna. Among these there were two cases accompanied by dislocation of the head of the radius, and a third with concomitant fracture of the neck of the radius. The dislocations were successfully reduced without arthrotomy, and had not shown any tendency to recur up to the time of consolidation of the ulnar fractures. The patients cannot be found now. During the same time there has been

only one isolated dislocation of the head of the radius; this also was successfully reduced and had not recurred one month later, when the patient was last seen.

That the combined injury is by no means rare, is proved by the large number of isolated case reports (119) collected by Stetten in 1908 (*Annals of Surgery*, 1908, xlviii, 275). To these may be added 20 previously unpublished cases included in Perrin's Thesis. Indeed the association of these injuries is so classical, that since the time of Malgaigne (1854) there has been a rule that in every case of fracture of the ulna alone, the existence of an anterior luxation of the radius should be suspected; and it may be added that in every case of anterior luxation of the radial head the surgeon should make very sure that no concomitant fracture of the ulna is overlooked.

As a matter of fact, while the fracture of the ulna usually is recognized, the luxation of the radius often is overlooked; and when swelling of the soft parts has subsided and the luxation is first noted, the surgeon should blame himself for neglecting the additional lesion. Modern text-books do not lay enough stress on this injury. It is true that the surgeon may argue that even had he recognized the luxation at first it might have been impossible to keep it reduced; and in this way he may console himself that no harm has been done. But in many cases harm will have been done, for the fracture of the ulna may unite with such deformity as to interfere very materially with subsequent treatment, or only fibrous union may result.

The association of nerve lesions, especially paralysis of the musculo-spiral nerve, has been studied with care by Stetten. He collected 9 such cases, and these, with 6 others mentioned by Perrin, make a total of 15 examples of nerve injury among a total of about 140 instances of the combined injury (luxation of the radial head and fracture of the ulna). Two of these cases of musculo-spiral paralysis appear to have developed immediately after the injury (Helferich, Albertin); in some

of the others the time of onset is not mentioned, but in most it developed late, being due to secondary neuritis from pressure on the nerve or from its being stretched over the displaced radial head.

It is therefore very evident that reduction of the luxation in recent cases is extremely important. If reduction is obtained, the ulnar fracture as a rule will heal in good position without further trouble. If, however, reduction of the dislocation is not obtained, the ulnar fragments will overlap or angulate, and union with deformity or non-union will result.

RECENT CASES WITH IRREDUCIBLE LUXATION.—If reduction in a recent case cannot be secured by manipulation alone, the surgeon should resort to arthrotomy; but he should be sure that reduction is impossible without. Perrin says Destot attempted reduction seven times, with the aid of chloroform, and only at the seventh attempt was successful. But Stetten warns, and I think rightly, of the danger of injury to the musculo-spiral nerve by too strong pressure on the radial head, and by hyperextension of the elbow. Perrin, moreover, would confine operative reduction in recent cases to patients over fifteen years of age; but as of 86 patients whose age is mentioned 52 were under fifteen years of age, this rule would exclude a large proportion of cases. The only reason for not operating on young patients is the expectation that the luxation will prove irreducible even after arthrotomy, and that excision of the head will be required, which of course would be undesirable before full growth was attained; but there seems no good reason why reduction should prove impossible in a recent case, if the operation is properly performed. In adults, I agree with Perrin that excision should be done for irreducible luxation, since here as elsewhere such a lesion is very apt to lead to dystrophic arthritis, which is painful and disabling.

The object of the operation is to remove the torn capsule from its obstructing position in front of the capitellum of the humerus, and from over the lesser sigmoid cavity of the ulna,

and to suture it around the neck of radius. It is said that usually the capsule is torn transversely above the head of the radius, while the orbicular ligament is split longitudinally; thus the tear in the capsule is T-shaped, and the inner triangular flap is blamed as the obstructing factor. Unless operation is done this flap is just pushed ahead of the radius, and no matter how long the head of the radius is held in proper position it will not stay there of itself.

Perrin collected five operations for recent injury, and I have been unable to find any published since the appearance of his Thesis, up to January 1, 1912. These cases are the following:

1. Le Dentu (1892): Case complicated by rupture of brachial artery; resection of radial head for irreducibility. Good motion recovered. Adult.

2. Delorme (1902): First operation, suture of ulnar fracture, and suture of head of radius to ulna. Dislocation recurred. Second operation, excision of radial head. Adult.

3. Durand (1909): Dislocation of radius reduced without operation; but fracture of ulna plated later. Adult. Perfect result.

4. Herman (1908): Case complicated also by fracture of neck of radius. Reduction of luxation by arthrotomy not permanent until the fracture of neck of radius was sutured. Then the luxation stayed reduced, and the ulnar fracture came into good position. Aged eight years.

5. Kirmisson (1902): Luxation irreducible by arthrotomy until ulna was sutured; this procured reduction of radius. Aged ten years. Examination four years later showed luxation had recurred; there was cubitus valgus of 166 degrees; flexion was incomplete, but the other movements were normal.

The results of these operations are not very encouraging, perhaps, but in the earlier operations there does not seem to have been very great effort made to suture the orbicular ligament around the neck of the radius, so as to prevent recurrence of the luxation.

In cases not complicated by ulnar fracture, operation in recent cases of anterior luxation of the head of the radius

appears to have been undertaken only four times; by Sprengel, Reerink, Schede, and Bardenheuer; and in every case arthrotomy without resection was successful, as it was in the case of an old dislocation without fracture reported by G. G. Davis (*Amer. Jour. of Orthop. Surg.*, 1911, viii, 586).

OLD CASES, WITH UNREDUCED LUXATION. Perrin recommends a delay of four or five months in adults, and longer in children, to see whether sufficient function will be secured without operation. Even in the best cases flexion and supination are limited; and in most cases the patients not only are unable to do any heavy work, but cannot even feed or shave themselves or brush their own hair with the injured arm.

Perrin collected 25 operations for old unreduced luxation of the head of the radius complicated by fracture of the ulna. To these may be added the operation by Kammerer, recorded by Stetten, and that by myself, which is the occasion of this paper.¹

These cases are as follows:

1. G. W. Norris (1843): Adult, non-union of ulna. Resection of ulna to secure end to end apposition. Infection. Only fibrous union.
2. Swinburne (1859): Aged eight years. Eight weeks after injury did osteoclasis for deformity of ulna; then reduced luxation, set fracture, and got excellent result.
3. Dorfler (1885): Osteoclasis, but luxation could not be completely reduced by manipulation. No age or end result.
4. Kirmisson (1889): Non-union of ulna, cured by insertion of ivory peg. Good union secured, but movements of elbow not improved (luxation not reduced). Adult.

¹ F. J. Cotton (Dislocations and Joint Fractures, Phila., 1910, pp. 236 and 295) mentions a case of old anterior dislocation of head of radius with ununited fracture of shaft of ulna. He reduced the luxation by arthrotomy and held the radius in place by a new ligament made of fascia and wired the ulnar fracture. Though bony union was not secured the dislocation remained reduced, and a useful arm was obtained. Woolsey (*Annals of Surgery*, 1912, i, 888) secured good function after plating the ulna, in one recent case; the dislocation of the radius was reduced without incision.

5. Gérard-Marchant (1890): Non-union of ulna. Suture of ulna; later resection of head of radius. Only fibrous union secured, but condition improved by operation.

6. Chevassu (1897): Ulna in good position. Head of radius excised. No attempt mentioned at reduction and capsulorrhaphy. Good result.

7. Albertin (1898): Paralysis of musculo-spiral nerve, treated by excision of head of radius. Good result.

8. Annequin (1898): Ulna in good position; head of radius excised. No attempt mentioned at reduction and capsulorrhaphy. Good result.

9. Gérard-Marchant (1898): Ulna in good position; head of radius excised. No mention of attempt at reduction and capsulorrhaphy. Good result.

10. Lejars (1898): For non-union of ulna. Ulna sutured by silver wire. Only fibrous union, but improvement.

11. Report by Perrin, operation by unknown surgeon in 1899. For non-union of ulna. Suture of ulna, and resection of head of radius. When seen by Perrin some years later, pseudarthrosis persisted, and state of patient was deplorable.

12. Tillmann (1901): Ulna in good position. Arthrotomy and suture of capsule. Luxation recurred.

13. Tillmann (1901): Same case as No. 12. Later excision of head of radius. Good result.

14. Schwartz (1902): Ulna in good position. Excision of head of radius. No mention of attempt at capsulorrhaphy. Good recovery.

15. Katzenstein (1903): Ulna in good position. Arthrotomy, reduction, and capsulorrhaphy. Aged eight years. Perfect functional result, but head of radius remained subluxated.

16. Loison (1903): Malunion of ulna. Osteotomy of ulna, and reduction of luxation by arthrotomy; no suture of capsule mentioned. Luxation recurred.

17. Loison (1903): Same case as No. 16. Later excision of head of radius. No operation on ulna this time, although union in bad position had again occurred. Poor result. Motion only from 90 to 120 degrees, rotation only half normal, and marked atrophy of limb.

18. Riese (1903): Ulna in good position. Arthrotomy and reduction; suture of capsule proved impossible. The end result gave great improvement, but head of radius still subluxated. Adult.

19. Bérard (1904): Non-union of ulna. Resection of head of radius, and modelling resection of lower fragment of ulna. No mention of attempt at reduction and capsulorrhaphy. Excellent result. Adult.

20. Zschock (1904): Non-union of ulna, and radial paralysis. Suture of ulna, and later excision of head of radius for persistence of radial paralysis. Adult. Good union in ulna, better elbow motion, no improvement in radial paralysis. No mention of attempt at reduction and capsulorrhaphy.

21. Legueu (1905): Excision of elbow for ankylosis following badly united fracture of olecranon with anterior dislocation of radius. Good result.

22. Capron (1906): Ulna in good position. Head of radius excised. No mention of attempt at reduction and capsulorrhaphy.

23. Tricot (1906): Ulna in good position. Reduction by arthrotomy. Capsule not sutured. Luxation recurred.

24. Tricot (1906): Same case as No. 23. Excision of head of radius for recurrence of dislocation. Good result.

25. Lambotte (1908): Malunion of ulna. Osteotomy of ulna, and excision of head of radius, as reduction proved impossible. Perfect result.

26. Kammerer (1908): Ulna in good position, musculo-spiral paralysis. Head of radius excised. Aged nineteen years. No attempt at reduction and capsulorrhaphy mentioned. Perfect recovery of power in three years, but luxation persisted.

27. Ashhurst (1911): See below.

These operations may be thus classified:

A. Fourteen cases in which the ulna had united in fairly good position:

Five treated by attempts at reduction of dislocation of radius. (In two of these the capsule was not sutured.) Luxation recurred in two cases (Tillmann, capsulorrhaphy done; Tricot, capsulorrhaphy not done). Good result in three cases (Katzenstein, Riese, Ashhurst; capsule sutured except in Riese's case).

Nine treated by excision of head of radius. In two cases (Tillmann, Tricot) for recurrence of luxation after reduction by arthrotomy; in two cases there was also musculospiral paralysis (Albertin, Kammerer). Good result in all.

B. Six cases in which ulna had united in bad position:

Two treated by osteoclasia of ulna and reduction of radius without arthrotomy (Swinburne, excellent result; Dorfler, result unknown).

One treated by osteotomy of ulna and reduction of radius by arthrotomy (Loison, luxation recurred).

One treated by osteotomy and excision of head (Lambotte, excellent result).

One treated by excision of elbow-joint, for ankylosis (Legueu, good result).

One treated by excision of radius, for recurrence of luxation (Loison, poor result).

C. Seven cases in which ulna was ununited:

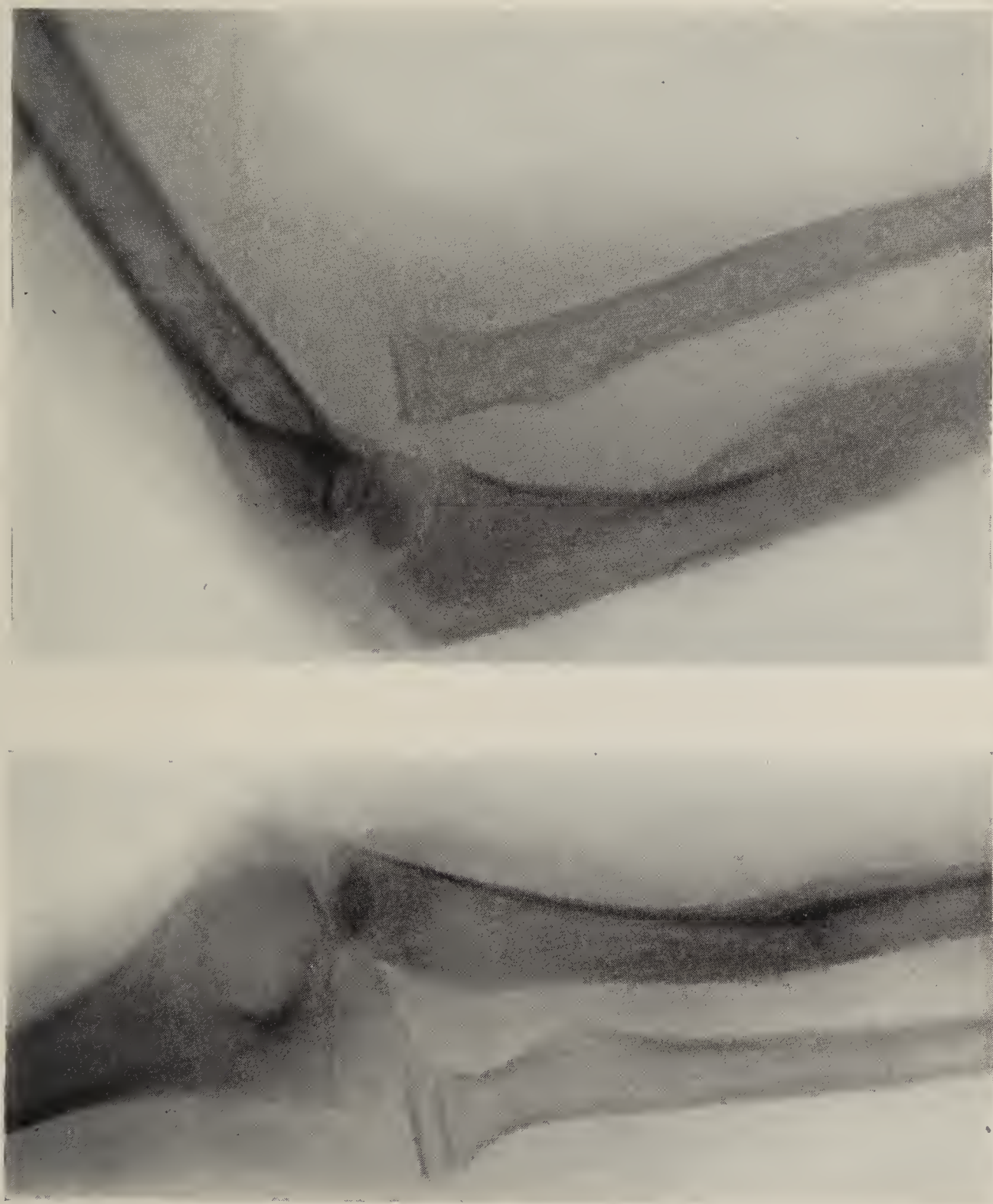
Three treated by operation on ulna, nothing done to radius (Norris, Kirmisson, Lejars, all improved).

Four treated by operation on ulna and excision of head of radius (Gerard-Marchant, with improvement; unknown surgeon, with bad result; Bérard, with excellent result; and Zschock, with improvement, but no relief of accompanying musculo-spiral paralysis).

CASE REPORT.—Thomas O'B., aged eleven years, in July, 1911, fell on the ulnar surface of his left forearm, causing a fracture of the ulna, at the junction of its middle and upper thirds; and by continuance of the force the head of the radius was dislocated forward. It does not appear that the existence of a dislocation was recognized. His arm was dressed on a splint, but he did not return to have his arm redressed until three weeks later. He then came under the care of Dr. Z. M. K. Fulton, my colleague at the Episcopal Hospital, and through his interest the patient was later referred to me for operation. At this time union in the ulna was fairly firm, there was not much deformity, but the dislocation could not be reduced. Four months after injury the boy was admitted to the Episcopal Hospital, in the service of Dr. Chas. H. Frazier. Figs. 74 and 75, from skiagraphs made at this time, show the condition of the bones. The ulna had united with slight angular deformity toward the extensor surface, and the head of the radius was luxated far forward. Flexion was stopped at 50 degrees by the radius butting against the shaft of the humerus; this

caused considerable pain. Extension was complete. The carrying angle was 160 degrees, that of the normal elbow being 170 degrees. Rotation of the forearm, as measured by the writer's instrument (*Amer. Jour. Med. Sci.*, 1912, i, 848), was possible through a range of 150 degrees (from 20 to 170 degrees); that is to say, there was a loss of at least 20 degrees of supination, and an increase in the range of pronation. The head of the radius was displaced upward and outward from its normal place, and was rather freely movable. There were no nerve lesions. Even momentary reduction was impossible; evidently the normal site of the radial head was filled up with soft tissues. There was a decided hollow in this location.

Operation (November 22, 1911).—No Esmarch band was used. An external incision was made, three inches and a half in length, along the supracondylar ridge and downward between the brachioradialis and the extensor carpi radialis longior. This interspace was opened and the head of the radius identified. The supinator brevis was partially detached from the neck of the radius, and pushed downward. The musculo-spiral nerve was displaced forward. The tendon of the forearm extensors was next dissected partially off the external lateral ligament of the elbow. The remains of the orbicular ligament were then plainly seen crushed against the capitellum of the humerus. At no time could there have been any possibility of reduction of the luxation without arthrotomy. The opening in the capsule was not large enough to readmit the head of the radius. The cup-shaped depression in the head of the radius was filled with granulation tissue, showing there had been some change in the cartilage. This granulation tissue was removed by sharp gouge forceps. The orbicular ligament was then raised from in front of the capitellum, it was incised in the long axis of the arm, and the head of the radius was replaced in its normal relation to the external condyle. The orbicular ligament was then carefully sutured with mattress sutures of chromic gut to the periosteum covering the neck of the radius, to the supinator brevis, and to the ulnar attachments of the external lateral ligament. From six to eight such sutures were inserted, and there was then no tendency for the radial head to relaxate. The supinator brevis was then sutured over the head of the radius; the extensor tendon was again attached to the external lateral ligament and the external condylar ridge; and the brachio-radialis was sutured to the extensor carpi radialis longior. The skin wound was closed with chromic catgut without



FIGS. 74 and 75.—Anterior and outward dislocation of head of radius, with fracture of shaft of ulna. Four months after injury.



FIG. 76.—Anterior and outward dislocation of head of radius, three months after reduction by arthrotomy and capsulorrhaphy. Compare with Fig. 75.

drainage. Not one ligature was required throughout the operation, which consumed an hour. The elbow was dressed in hyperflexion.

Some infection of the skin sutures occurred, but the deeper structures healed perfectly. The elbow was kept in hyperflexion for about five weeks, to give the orbicular ligament plenty of time to cicatrize. Rotation of the forearm was practised at each dressing, and never was painful.

The further progress of the case was slow, as regards the return of extension. Fig. 76 shows the elbow region three months after operation. Full extension was not regained for six months after operation, being aided by gentle massage and passive motion, which were instituted first three months after operation.

At present, over six months since operation,¹ the head of the radius retains its normal site in all positions of the elbow; and it cannot be forced into subluxation even by direct pressure. Flexion is present to 40 or 45 degrees, there is complete extension, the carrying angle has been restored to normal, and the arm feels much stronger than before the dislocation was reduced. There is no palpable evidence of the ulnar fracture. The result may be considered nearly perfect.

From this study, I think certain rational conclusions may be drawn:

In *recent cases* secure reduction of the dislocated radial head, by arthrotomy and capsulorrhaphy if necessary. As Kirmisson says, in such injuries, the dislocation is everything, the fracture is nothing. When reduction is secured the fracture almost invariably will fall into good position. If it does not, it may be fixed by suture or plate.

In *old cases with the ulna united* attempt reduction of the dislocation by arthrotomy and retain the radius in place by capsulorrhaphy. If reduction after arthrotomy proves impossible, as it may if the ulna has united in bad position, it is better to excise the head of the radius than to interfere with the ulna, unless the deformity in the ulna is very extreme. In such cases osteotomy of the ulna may be done.

¹ Now, when reading proof, over nine months since operation, function is perfect.

In *old cases with non-union of the ulna* expose the ulnar fracture first, and after freeing the fragments, secure reduction of the dislocation (by arthrotomy if necessary, including capsulorrhaphy); then treat the ulnar fracture as if no dislocation had existed.

In *cases with musculo-spiral paralysis* excision of the radial head failed to secure a good result in one case (Zschock), and there is no evidence that reduction and capsulorrhaphy would not have been successful in two others (Albertin, Kammerer) in which excision was done.

OPERATIONS FOR OLD FRACTURES OF THE ELBOW¹

BY ASTLEY P. C. ASHHURST, M.D.
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1. *Arthroplastic operation for old fracture of external condyle of humerus, with gunstock deformity.* James W., aged five years, broke his right elbow in July, 1908. When he came under Dr. Ashhurst's care in October, 1908, flexion was possible only to 50 degrees and extension only to 145 degrees. There was cubitus varus, the forearm making an angle of about 200 degrees with the arm. Skiagraph showed fracture with outward rotation of the external condyle. For six weeks subsequently light massage and passive motion were employed, with the result that motion was increased 10 degrees in flexion, the range now being from 40 to 145 degrees.

On November 18, 1908, Dr. Ashhurst operated, in the Episcopal Hospital, service of Dr. C. H. Frazier. The joint was opened by an external longitudinal incision. A little granulation tissue, springing from the humerus and inside the joint cavity, was excised. The external condyle was found to be displaced down and back, as well as rotated. Enough of this was removed to permit full extension of the elbow, as well as to overcome the cubitus varus. The olecranon fossa on the posterior surface of the humerus was also deepened by curette, so as to admit the olecranon in full extension. A cross incision was then made in the skin, backward across the arm from the upper extremity of the longitudinal incision, and a flap of fat was dissected up, with its base below; this fatty flap was then inverted over the surface of the external condyle, all the cartilage of which had been removed down to the bony centre for the capitellum. The fatty flap was sutured in place with chromic gut, and this was used for

¹ Read before the Philadelphia Academy of Surgery May 6, 1912. Reprinted from *Annals of Surgery*, October, 1912.

repairing the external lateral ligament and for closing the skin. The elbow was dressed in hyperflexion. The time of the operation was fifty minutes.

The first dressing was made ten days later, when the wound was found healed, and all the skin sutures absorbed. Motion was free and painless from 45 to 90 degrees. The arm was now carried in a sling. On December 5, there was motion from 40 to 140 degrees.

At present, three and half years after operation, there is perfect function, no varus deformity, full flexion, but extension only to 150 degrees.

2. *Arthrotomy and suture of fragment for ununited fracture of external condyle of humerus.* Mary G. fractured her right elbow when two years old. At the age of nine years she applied to Dr. Ashhurst for deformity and slight disability from weakness of the joint. Examination showed an ununited fracture of the external condyle, which moved with the head of the radius in flexion and extension movements, which were not limited. The condyle was freely movable on the shaft of the humerus antero-posteriorly. In full extension there was cubitus valgus of 160 degrees; and this could be increased to 140 degrees by abduction of the forearm, which caused ascent of the external condyle on the shaft of the humerus. A skiagraph showed an ununited fracture of the external condyle, with rotation of the fragment anteriorly and outward, around a longitudinal axis.

The girl was admitted to the Episcopal Hospital, in the service of Dr. C. H. Frazier, and Dr. Ashhurst operated on October 23, 1909.

An incision four inches long was made on the outer side of the joint, between the supinator longus (brachioradialis) and the extensor muscles; the radial nerve was displaced anteriorly with the brachioradialis, and the humerus was bared. The elbow-joint was then opened through the line of fracture, and fibrous union was found. The humeral side of the fracture was chiselled off until healthy bone was found, and an attempt was made to rotate the condyle inward and backward, to restore normal relations. To accomplish this most of its attachments had to be severed, but the external lateral ligament passing from it to the orbicular ligament of the radius was carefully preserved. It was impossible to secure very accurate apposition. Then the fractured surface of the condyle was chiselled off. Best apposition was obtained with the elbow in full extension. The condyle was then sutured to the shaft with three chromic gut sutures, one of which passed

through bone on both sides (shaft of humerus and centre of capitellum), and the others through cartilage and periosteum. The deep fascia was closed with chromic gut sutures and the skin with silkworm gut. A few strands of plain catgut were inserted as a drain. The arm was dressed in full extension, with the forearm in full supination, on a straight anterior splint, the carrying angle being about 170 degrees. The time of the operation was seventy minutes.

The first dressing was made ten days later (November 2, 1909). The catgut drain had been absorbed, the skin incision was healed, and the silkworm gut skin sutures were removed. Rotation in the forearm was normal in extent and painless; flexion to about 160 degrees was made without pain. The arm was again dressed in full extension. A week later the elbow was dressed at an angle of 135 degrees, flexion beyond which was painful. The fragment seemed firm, but in full extension there was still a little lateral mobility. Progress thereafter was uneventful.

At present, two and a half years after operation, there is perfect function, flexion and extension being complete as before operation. There is no more lateral mobility in the joint than in the normal left elbow, the external condyle is firmly united to the humerus, and the only deformity is slight increase in cubitus valgus, which is 150 degrees, the normal carrying angle in this girl being 170 degrees. This may be remedied by an osteotomy of the humerus above the condyles.

3. *Old supracondylar fracture of humerus, with inward displacement of lower fragment, giving gunstock deformity.* James L., a boy aged seven years, on July 26, 1911, fell over backward, crushing his left elbow between his back and the ground, his arm being held across his back at the time. The physician who was consulted dressed the elbow in a flexed position, probably corresponding to the "Velpeau" position. That night the father removed all the bandages because the arm was painful. The next day another physician dressed the elbow. Three weeks later the child was first seen by Dr. Ashhurst. The elbow at that time was dressed at an angle of 80 degrees, and was held against the chest by adhesive plaster. Motion was limited and painful. There was a sharp projection of bone in the region of the external condyle, and the skin overlying this was on the point of sloughing. On account of the swelling the exact relation of the bones could not be determined. The elbow was kept quiet until all inflammatory symptoms had subsided.

Skiagraphs made September 19, 1911 (Figs. 77 and 78), showed a supra-condylar fracture, with marked internal displacement of the lower fragment; the sharp bony projection was caused by the lower end of the upper fragment jutting into the soft parts above the external condyle. A photograph made from behind, with the elbow flexed to a right angle and the bony points marked in ink, shows the deformity very well (Fig. 80). By October 27, three months after injury, the range of motion was still limited to 35 degrees of flexion and 140 degrees of extension. The carrying angle was lost, there being cubitus varus of 200 degrees (*i. e.*, the forearm was 20 degrees to the inner side of the axis of the arm, instead of about 10 degrees to the outside). There being no prospect of further improvement without operation, the patient was admitted to the Episcopal Hospital, in Dr. C. H. Frazier's service, and was operated on by Dr. Ashhurst before the members of the Congress of Surgeons of North America, November 10, 1911.

Operation: An incision four inches long was made on the posterior surface of the elbow, splitting the triceps muscle down to the shaft of the humerus. The periosteum was stripped away from the shaft on both sides of the incision, and *all around the shaft*, carrying the ulnar nerve well out of harm's way. A smooth retractor was then passed around the humerus, to protect the soft parts on the anterior aspect of the joint. The triangle of new formed subperiosteal bone, above the internal condyle, was then excised. Then the humerus was cut across in the line of the old fracture, just above the condyles, the section being made by osteotome and mallet. The lower fragment was then pushed forward. The elbow-joint had not been opened. Then, with Hey's saw, one-eighth of an inch of the lower end of the diaphysis was sawed off, and by means of gouge forceps and Liston's bone forceps the lower end of the diaphysis was trimmed until it fitted the elbow fragment of the humerus. This lower fragment was very small, extending only from the level of the olecranon fossa down into the joint, and nothing could have been removed from it without entering the joint, which it was not desired to do. The transverse axis of the upper fragment was made such that when fitted to the joint fragment the carrying angle was restored. It was then ascertained that both in hyperflexion and in extension to 170 degrees the lower fragment held its normal relation to the upper fragment without bone suture. The wound was then closed by interrupted chromic catgut sutures in layers, as follows: (1)



FIGS. 77 and 78.—Old supracondylar fracture of humerus, inward displacement of lower fragment producing cubitus varus. (Case III, before operation.)

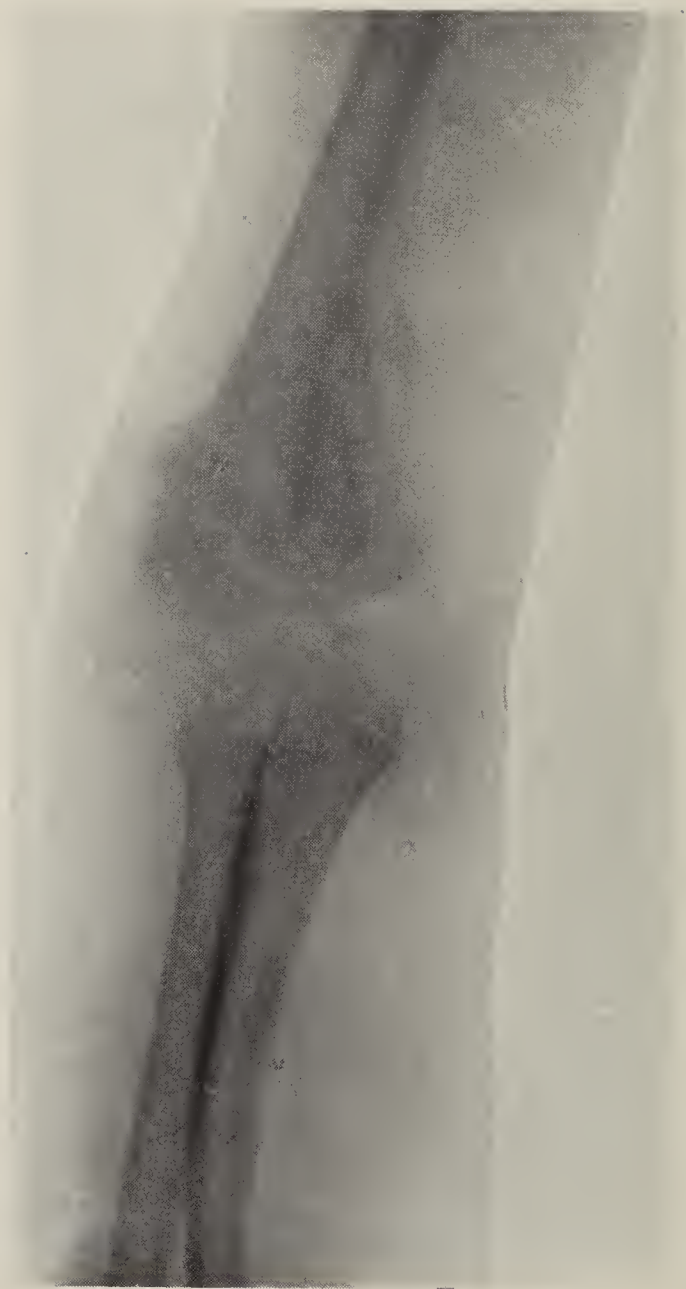


FIG. 79.—Case III. Four months after operation.



FIG. 80.—Old supracondylar fracture of left humerus, inward displacement of lower fragment. (Case III.)



FIG. 81.—Same patient six months after operation. Dots on condyles and olecranon. (Case III.)

periosteum; (2) deep fascia over triceps; (3) skin. No drain. After the wound was sutured, the fracture was reduced (by hyperextension, direct traction, and subsequent hyperflexion) just as if it was a recent injury, with no wound present. The elbow was dressed in hyperflexion (20 degrees), the forearm being brought up upon the arm directly in the sagittal plane. The wound was then dressed, and the elbow maintained in hyperflexion by the usual bandage. The time of the operation was thirty-five minutes.

A skiagraph (lateral view) made four days later showed that the lower fragment was tilted a little forward, so the hyperflexion was slightly reduced at the first dressing, November 15.

There was some infection of the skin sutures, but otherwise healing was uneventful and entirely painless. After three weeks the arm was carried in a sling, and soon after this active use was encouraged.

As extension was slow in returning, a light brace with a Stromeier screw was applied, at the suggestion of Dr. G. G. Davis, some three months after operation. This was worn at night, and in a few weeks full extension could be secured every evening, very soon after the brace was put on, but during the day when it was not worn voluntary extension was somewhat limited.

At present, six months after operation, there is easy voluntary extension to 160 degrees, flexion to 35 degrees, a normal carrying angle, and scarcely any visible deformity (Figs. 79 and 81).

A REPORT OF TWO CASES OF BONE CYSTS¹

BY LOUIS H. MUTSCHLER, M.D.
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THESE two cases came under my observation during my service at the Episcopal Hospital, in 1912.

CASE I.—F. H., white, male, aged fifteen years, born in United States. Admitted to the surgical ward, February 26, 1912; discharged April 27, 1912.

Family History. No tuberculosis, no tumors of any description.

Personal History. Has had measles, otherwise has always been well. Gave occupation as messenger boy.

One year ago had the upper end of the right humerus fractured. Ten days before admission to the hospital fell down stairs striking the upper part of the right arm. This was followed by some slight soreness and swelling.

Examination. He is well nourished and of fair development; the submaxillary glands of both sides are slightly enlarged, no other glandular enlargement. There is a fusiform enlargement, about the size of a hen's egg, of the upper end of the right humerus. There is very little tenderness, no redness or elevation of temperature; the skin is not adherent; motion in the shoulder-joint is normal. The right arm, at the seat of growth, measures nine and one-eighth inches in circumference and the left arm eight and five-eighths inches at a corresponding point. I was unable to elicit any crepitus or preternatural mobility. An x-ray picture was taken and showed enlargement of the upper end of the humerus, at and below the epiphyseal line, with rarefaction of the bone partly extending into the head of the humerus. No fracture is seen.

¹ Read before the Episcopal Hospital Clinical Society, March 17, 1913.



FIG. 82.—Bone cyst of right humerus. Before operation. Case I.

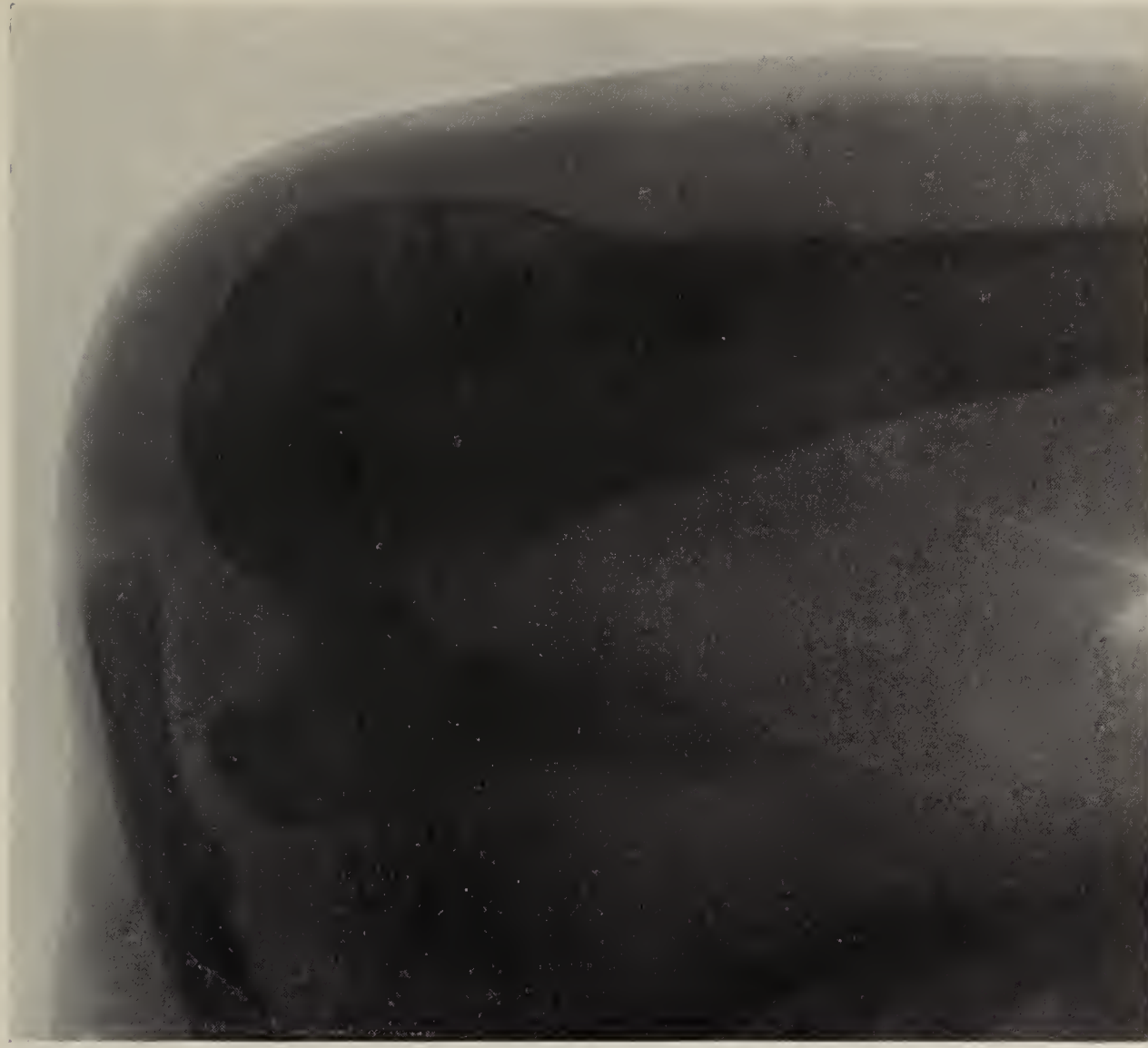


FIG. 83.—Bone cyst of right humerus, one year after operation. Case I.



FIG. 84.—Bone cyst of right tibia, anteroposterior view. Case II.

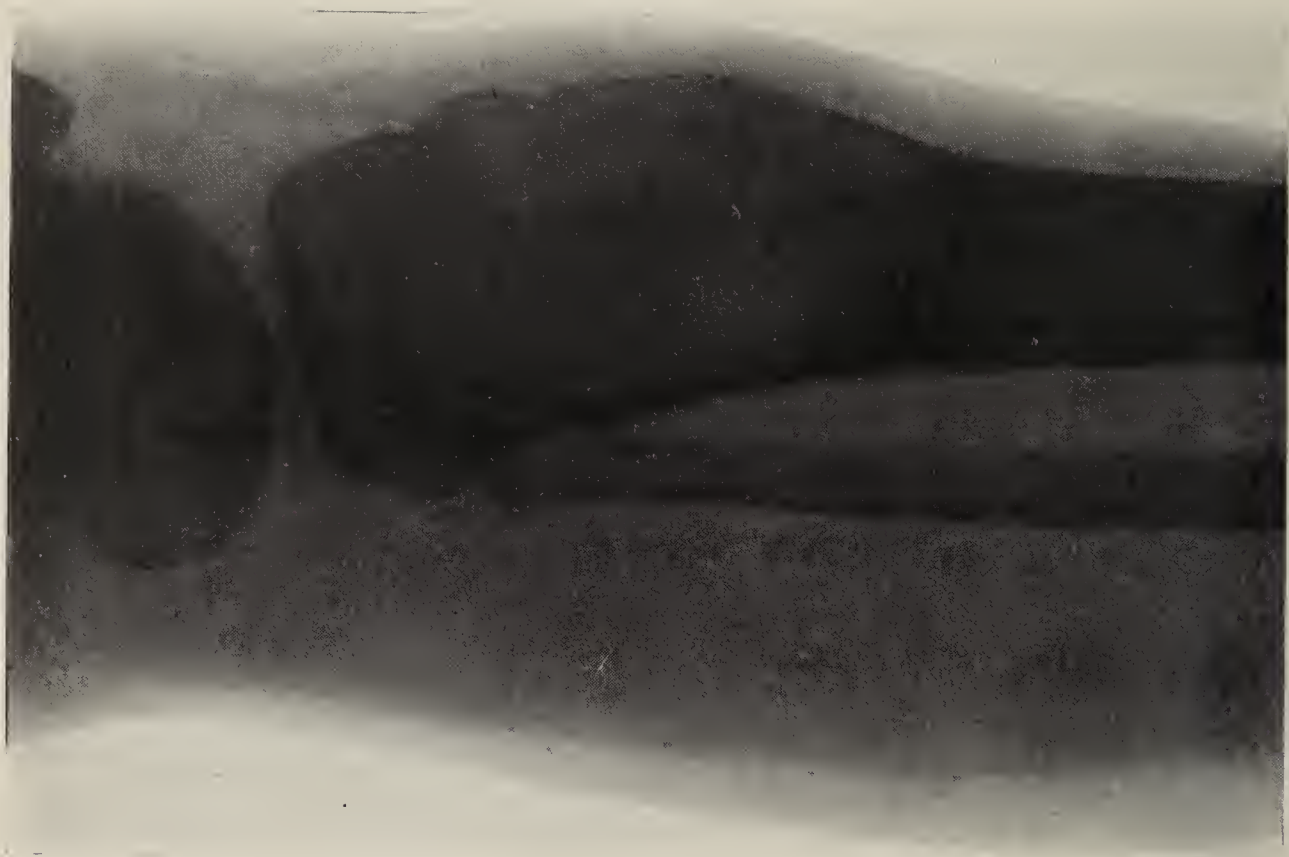


FIG. 85.—Bone cyst of right tibia, lateral view. Case II.

Operation. March 8, 1912. A vertical incision, about two and a half inches long, was made through the fibres of the deltoid muscle and the bone was exposed. The tumor proved to be a cyst having a wall not thicker than an egg shell, which was easily broken through, revealing a clear and almost colorless fluid of the consistency of water. The cavity extended to the head of the bone and did not have a lining membrane. The cavity was as large as a good sized hen's egg.

The cavity was lightly curetted and packed with gauze. The incision was partly closed with stitches. The cavity gradually filled in and the patient was discharged recovered.

A Wassermann test was made and proved to be negative. Smears and cultures taken from the fluid were negative for bacteria. The smear showed a very few polymorphonuclear leukocytes and lymphoid cells.

This patient's mother told me a few days ago that her son was entirely well.

CASE II.—H. D., white, female, aged twelve years, born in United States. Occupation, school. Was admitted June 10, 1912, discharged August 21, 1912.

Family History. Father died of carcinoma of the stomach; mother living and well; one sister died of consumption.

Personal History. Has had measles and chorea, otherwise has been well.

Present Illness. Three months before admission while going up stairs struck her right knee. She did not have much pain or swelling at the time but there has been a slow and progressive enlargement of the leg immediately below the knee. There has been very little pain or tenderness and only slight redness. The leg had been treated by an osteopath.

Examination. The patient was well nourished and I was unable to find any enlarged glands. The upper end of the right tibia is enlarged. There is no redness or elevation of temperature; the skin is not adherent; there is no pain and only slight tenderness. The growth is spindle-shaped and seems to be confined to the upper end of the tibia. Motion in the knee-joint is normal. An x-ray picture was taken and showed an enlargement of the upper end of the tibia with rarefaction of the bone extending from the epiphyseal line downward.

Operation. June 25, 1912. An incision was made over the growth and the bone was found to be soft, and very thin. The wall was readily penetrated, revealing a quantity of slightly brownish, bloody fluid.

The wall of the cyst seemed to have a jelly-like lining and felt like clotted blood. The cavity was rubbed with dry gauze and then packed with iodoform gauze. The incision was then partly closed and the patient made a good recovery.

The Wassermann test was taken and proved negative.

I have indirectly heard from this patient in the last few days and she is apparently entirely well.

In examining the records of the Episcopal Hospital I have been unable to find more than four cases of bone-cyst recorded, two of which were mine, another occurring under the service of Dr. Neilson and the fourth under the service of Dr. Frazier and reported by Dr. Ashhurst.

Dr. Neilson's case occurred in a white female, aged eighteen years. She was admitted March 17, 1910, and discharged April 26, 1910. The diagnosis on admission was fractured shaft of the left fibula. She gave the following history. On the day previous to her admission while running she turned suddenly and fell, hurting her left leg. This was followed by moderate pain and slight swelling. There was no crepitus or deformity. An x-ray picture showed rarefaction of bone, about one and a half inches in extent, one inch from the upper end of the left fibula. She did not remember ever having injured this leg before. The previous summer she had fallen from a car but fell on her right side.

March 26, an incision was made over the upper part of the left fibula and a cavity about the size of an English walnut and filled with clotted blood was discovered. The walls of the cavity were curetted and the cavity packed with gauze. The patient was discharged recovered.

The case occurring under Dr. Frazier's service, and operated upon by Dr. Ashhurst, was a white male, aged thirteen years. He was admitted January 7, 1913, and discharged January 22, 1913. He had a cyst of the right humerus. There was a history of an accident which had occurred two years previously and when the patient was admitted to the hospital an x-ray picture showed a recent fracture with a cyst of the right humerus. This case has already been reported before this Society by Dr. Ashhurst.

Although probably not more than a hundred cases of benign bone cysts have been reported and the literature upon

the subject is not extensive, I believe the condition is far more common than generally supposed. Bloodgood, in an able paper presented before the American Surgical Association, May, 1910, reports a study of 89 cases, which he had been able to collect up to that time. I have been able to find the reports of 5 additional cases not including the 4 treated at the Episcopal Hospital.

Many modern books on surgery fail to describe or even mention cysts of bone, although treating of bone tumors and cysts of other parts of the body.

Cysts of the jaw bone, known as dentigerous cysts, due to misplaced or unerupted teeth, are not uncommon and are not to be considered with these cysts of the long bones.

Some of the early writers claimed that bone cysts were not primary but resulted from the degeneration of previously existing growths. As early as 1834 Dupuytren distinguished between solid and fluid tumors of the bone.

In 1876, Virchow studied a bone-cyst which was discovered at autopsy and he concluded from his observation of this one case that these cysts usually resulted from the degeneration of chondromas arising from the misplaced bit of epiphyseal cartilage.

More recent study and investigation of this class of cysts seems to indicate that traumatism plays an important part as an etiological factor.

There is a distinct history of traumatism in the two cases which I am reporting, also in the case reported by Ashhurst, but in the case treated by Neilson there was no positive history of a previous injury.

The etiology of bone cysts, however, is still very obscure. Bloodgood classifies benign cysts into two groups as follows: (a) True bone cysts, which have a definite relation to osteitis fibrosa, and (b) cysts in the medullary cavity due to other conditions.

The study of these cases shows that with a few exceptions bone cysts occur in the youthful individual. In the four

cases treated at the Episcopal Hospital all were under nineteen years of age.

As you know, at this age the head of the long bones is still separated from the shaft by the epiphyseal cartilage, and as these cysts occur, as a rule, at the end of the long bones they may have their origin in the cartilage.

Most cases occur in the long bones of the arm and leg, especially the humerus, femur, and tibia, but a few have been reported where the long bones of the hand and foot have been involved. The symptoms in benign bone cysts are not pronounced in character and are slow in making their appearance. The patients are usually healthy individuals.

The first symptoms are pain and swelling and, with a gradual absorption of the bone, fractures occur after slight injuries and often are the first intimation of the existence of a cyst. As the symptoms are slow in making their appearance the cysts may have existed for some time before the objective symptoms occur. Pain is not a constant symptom and when present is usually of a mild type. Tenderness as a rule is absent; in both of my cases there was very little pain or tenderness, but swelling was pronounced. The swelling is of fusiform character and is regular in outline. The skin and soft tissues are not adherent and there is no redness nor elevation of temperature. In a cyst with thin walls crepitation, resembling the cracking of an egg-shell, may be elicited; this is especially true where a fracture has occurred.

Bone cysts may be easily confused with a number of different bone diseases, such as periostitis, chronic abscess, old fractures, osteitis, etc. The most important point, however, is to differentiate between these solitary bone cysts and malignant tumors.

Since the use of x-rays the surgeon is materially aided in his diagnosis of bone diseases. It is quite probable that before the discovery of Röntgen rays limbs were unnecessarily sacrificed through a mistaken diagnosis of simple bone cysts for malignant tumors. In the instance of the two cases which

I am reporting, Case I was admitted as sarcoma of the humerus and Case II as osteomyelitis of the right leg.

The youthful age at which these growths appear, their slow development, their uniform ovoid shape, the lack of pronounced symptoms, such as pain, tenderness, redness, elevation of temperature and the soft tissues not adhering, the absence of any enlarged lymph glands and no impairment of the general health are symptoms which should help in making a diagnosis. In those cases where an *x*-ray has shown a rarefaction of the bone tissue, aspiration would be an additional aid in determining the nature of the condition.

The treatment of these bone cysts is simple. The cyst is opened and the contents evacuated after which the cyst wall is lightly curetted and the cavity loosely filled with gauze. Recovery, as a rule, is rapid.

CASE OF BONE CYST INVOLVING THE UPPER END OF THE HUMERUS¹

BY ASTLEY P. C. ASHHURST, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

JOSEPH R., aged twelve years, fell and injured his right shoulder in the summer of 1911. He was treated for a sprain. He fell again in November, 1911, and broke his humerus. About ten days after this injury he was admitted to Dr. Frazier's service in this hospital, and came under my care. The appearance of the arm is well shown in the photograph (Fig. 86); there evidently was an unreduced fracture of the upper end of the humerus. The outlines of the fragments were indicated in ink before the photograph was made. A skiagraph (Fig. 87) confirmed the diagnosis; nothing else abnormal was noted at the time. Excellent reduction, as shown by another skiagraph, was secured under a general anæsthetic, and recovery of perfect function was uneventful.

About three months later (March, 1912), another skiagraph (Fig. 88) was made, to see if there had been any permanent injury to the epiphyseal cartilage. There were now indubitable evidences of a change in the upper portion of the medulla of the humerus: the whole upper end of the diaphysis was thickened; two rarefied areas in the medulla were surrounded by denser bone; and there were indications of bony trabeculæ crossing the rarefied areas. The functions of the joint were perfect, there was absolutely no deformity, and there were no subjective symptoms of any kind. For these reasons no treatment was instituted, but the boy was kept under observation.

In August, 1912, nine months after the injury, another skiagraph was made (Fig. 89); this showed the cystic change more developed, with thinning of the cortex. There were absolutely no subjective

¹ Read before the Episcopal Hospital Clinical Society, September 16, 1912.



FIG. 86.—Photograph of original fracture of the upper end of the humerus, before reduction. December 8, 1911.

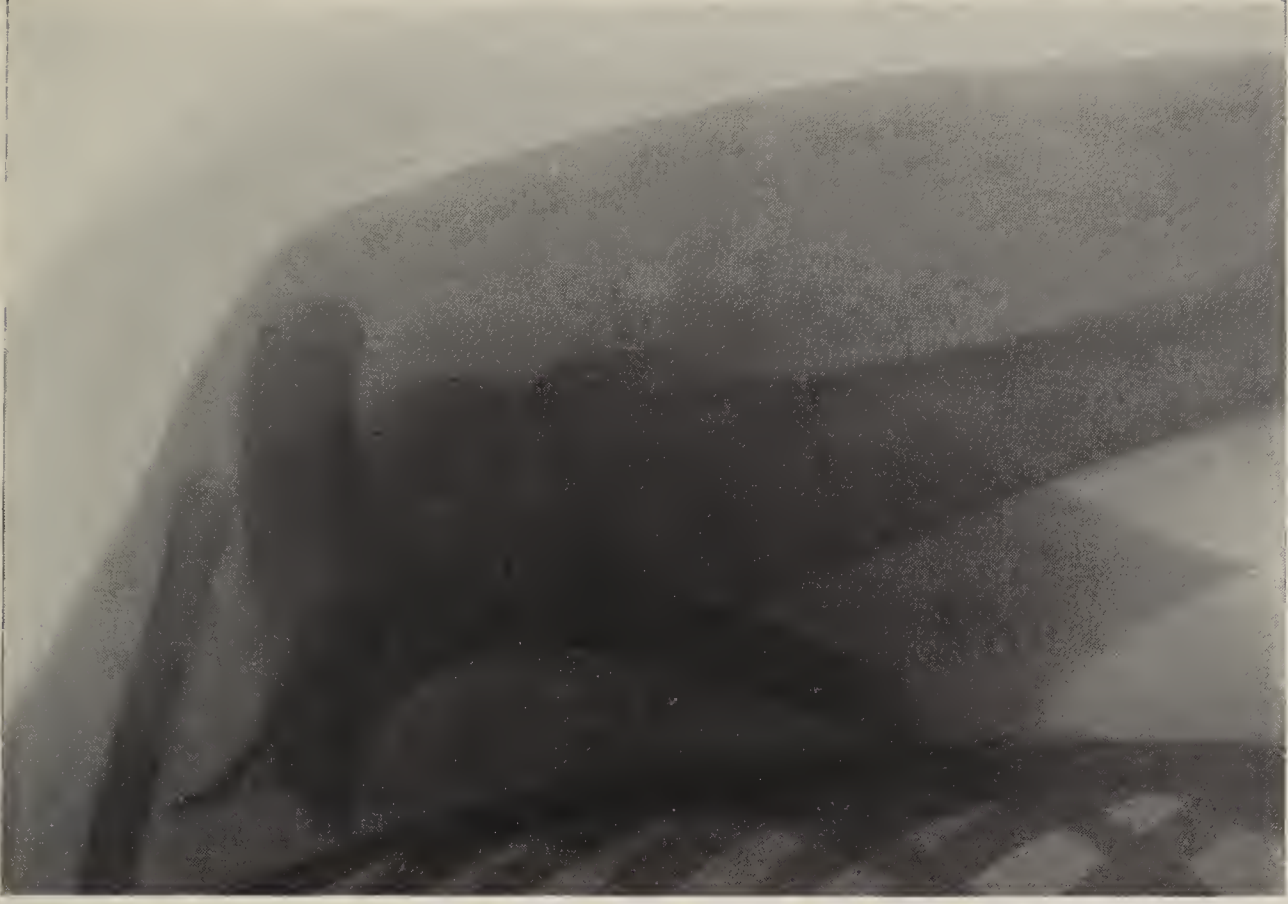


FIG. 87.—Fracture of upper end of right humerus. Skiagraph December 8, 1911.

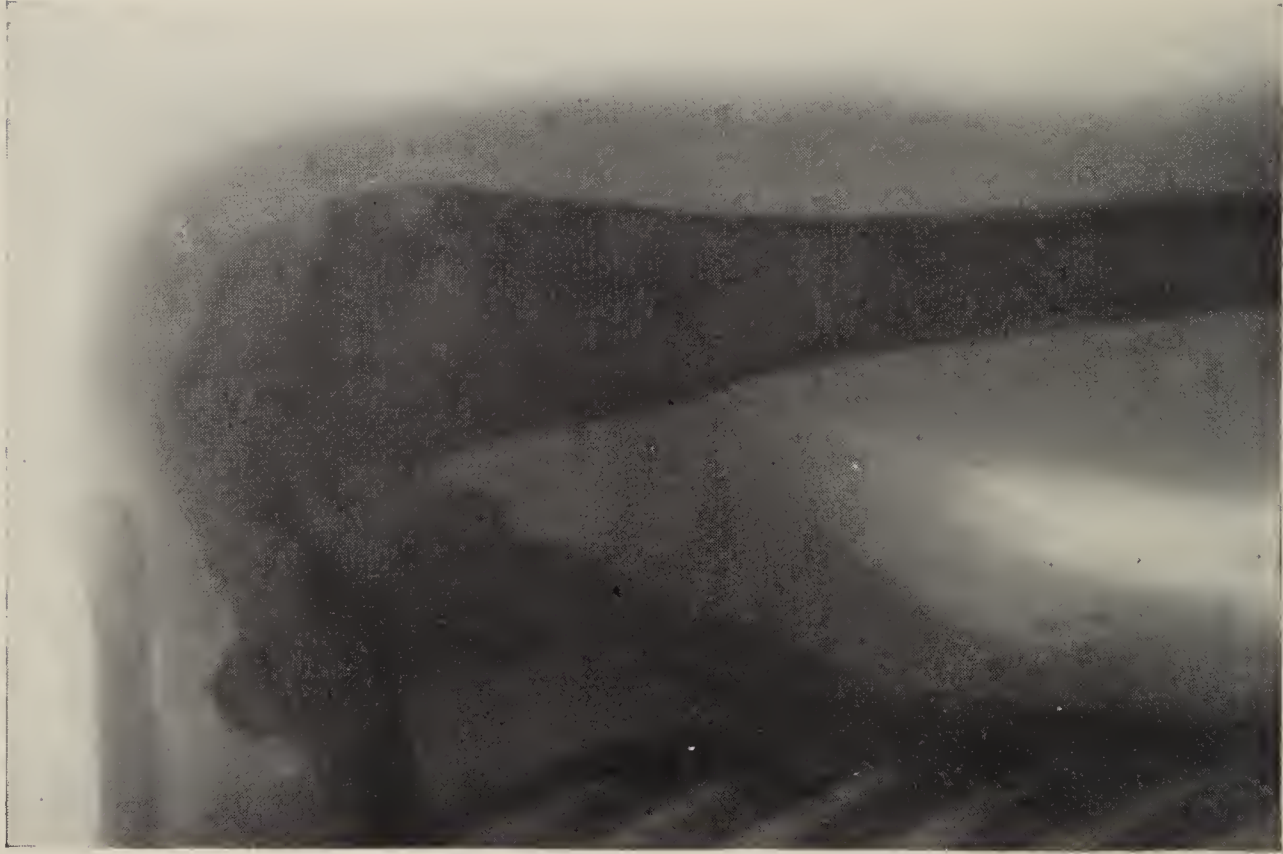


FIG. 88.—Bone cyst of humerus, three months after fracture.
Skiagraph made March 26, 1912.



FIG. 89.—Bone cyst of humerus, nine months after fracture.
Skiagraph made August 15, 1912.

symptoms whatever, and all the functions of the shoulder joint, including external rotation and abduction, were fully preserved. On palpation, however, a distinct though slight thickening of the humerus could be detected, on comparison with the other arm.

These are interesting cases, and the questions of *diagnosis* and *treatment* do not appear to be entirely settled. Looking more critically at the first skiagraph taken (Fig. 87), it can be seen that even at that date there was some pathological change other than the fracture; and it appears probable that the latter lesion, as the result of not very severe trauma, was predisposed to by the pre-existing pathological changes in the bone. In such young children cystic changes in bone are almost always benign in character, and the condition in this patient probably is of this nature. The relation of the so-called fibrous osteitis to the development of benign bone cysts is not well understood, but it seems not unlikely that the cyst is only a more advanced stage of the same process. Myeloma (formerly called giant-celled sarcoma) is quite rare in young children, being most common in adolescents and young adults; but its relatively benign character places it pathologically much closer to benign bone cysts than to the sarcomas of bone.

Operative treatment I believe is indicated in the present case as the condition evidently is progressive; but it has been refused by the child's parents, and in view of the perfect function and the entire freedom from subjective symptoms, it has not been urged very insistently. One or two cases are on record where a benign bone cyst retrogressed spontaneously but the lesion probably was not so far advanced as in this case.

In undertaking an operation such as this, temporary hæmorrhage by the Esmarch band is almost indispensable. In some cases where no such method was employed hemorrhage during the operation has been alarming. In the present case the band would have to be applied as for an amputation at the shoulder. The cyst should then be opened, and its con-

tents removed. In benign bone cysts the contents are blood-stained fluid; in myeloma the contents resemble currant jelly, but may have white patches resembling those seen in cases of osteitis fibrosa. The cyst wall should be thoroughly removed by gouge or Volkmann's sharp spoon, so as to prevent recurrence. Metastasis is unknown. The bony cortex should be preserved if possible. Hence the advantage of doing the operation before the cortex is destroyed. If the cortex can be preserved, and the cavity is not very large, it should be allowed to fill with blood clot, or could be packed with iodoform gauze; better still, it could be filled with Mosetig-Moorhoff's iodoform bone wax, and the soft parts closed without drainage. If the cavity left after evacuation of the growth is too large to make one of these courses desirable, the cortex can be crushed in, so as to obliterate the dead space. As a last resort, and in cases where it is impossible to leave behind enough of the cortex to preserve the form of the bone, the gap may be filled by a transplant taken preferably from the patient's own tibia.

Postscript. The boy fell again on January 6, 1913, and injured his right shoulder. He came at once to the Episcopal Hospital and was seen by me. A skiagraph (Fig. 90) showed a partial fracture of the cyst wall below the greater tuberosity. The enlargement of the upper end of the humerus is shown in the photograph (Fig. 92) made at this time. The parents now consented to operation.

Operation by Dr. Ashhurst, January 10, 1913. The patient was etherized and an Esmarch band was applied above Wyeth's pins, as for shoulder-joint amputation. An incision was made from the coracoid process down to the insertion of the deltoid muscle along the interspace between the deltoid and pectoralis major. The cephalic vein was left attached to the deltoid. Sterile gauze was then sutured to the skin margins of the wound. Then with another scalpel the intermuscular incision was deepened and the periosteum was incised just to the outer side of the long tendon of the biceps. The periosteum was thickened, but no distinct new subperiosteal bone was present. The fracture on the outer side of the surgical neck was thus exposed; one shell

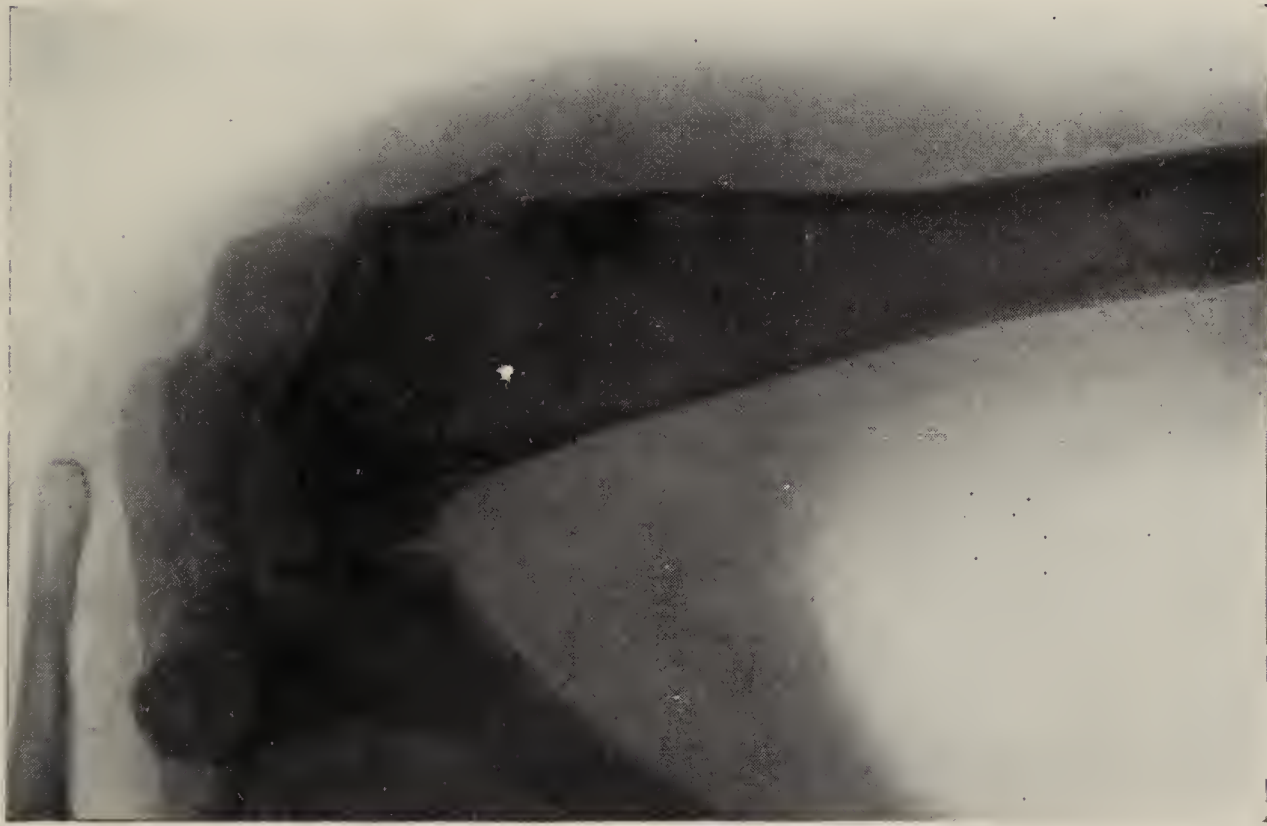


FIG. 90.—Fracture of bone cyst of humerus. Skiagraph made January 6, 1913, a few days before operation.

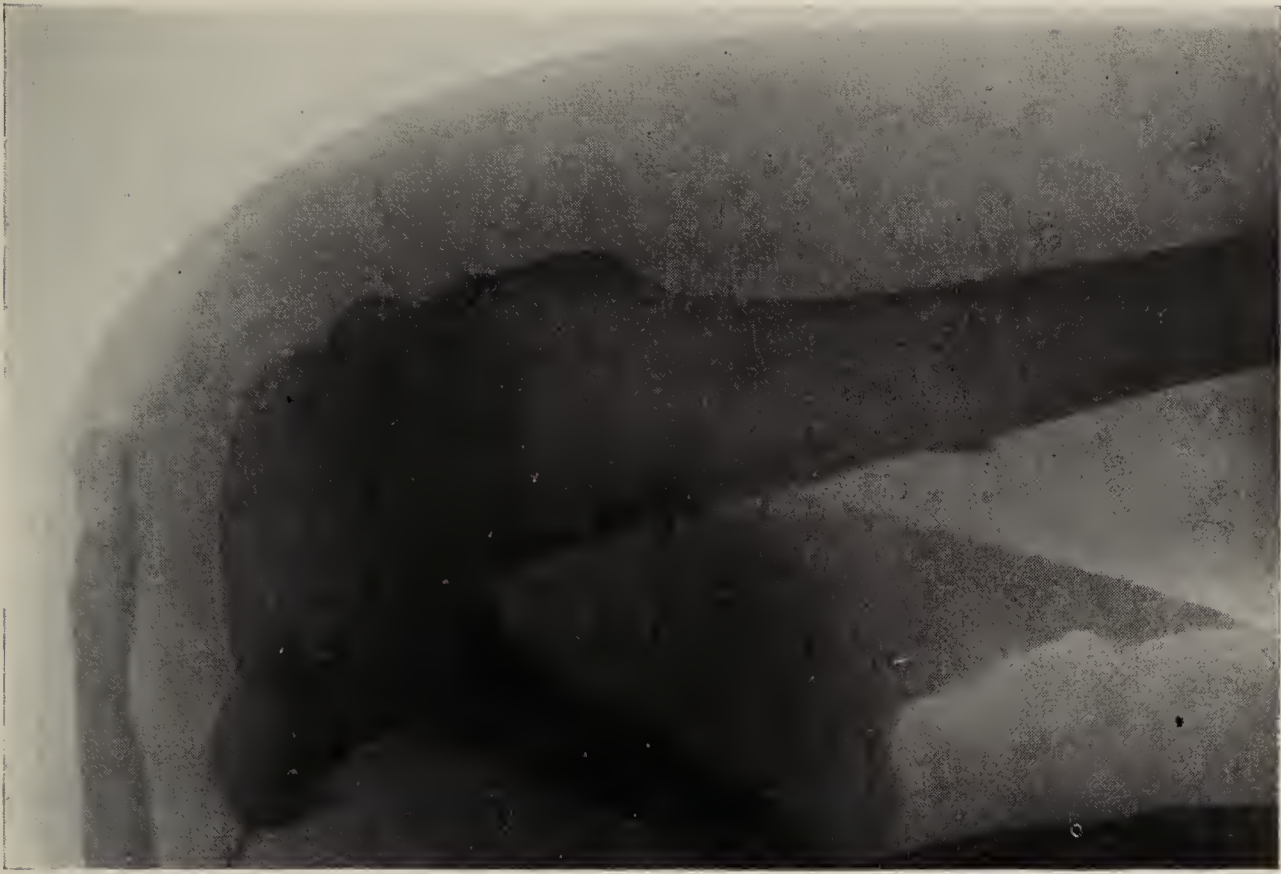


FIG. 91.—Bone cyst of humerus. Skiagraph made January 11, 1913, just after operation.



FIG. 92.—Photograph made January 7, 1913, three days before operation. Visible enlargement of upper end of humerus.



FIG. 93.—Photograph made March 10, 1913, two months after operation. Cicatrix accentuated purposely in reproduction.

of the cortex, as shown in the skiagraph, stood out from the shaft, and from this there was a faint line of fracture running transversely inward. No rotation of the lower fragment on the upper was possible.

The cortex, which was about 2 to 3 mm. thick, was then cut away with gouge, exposing the medullary cavity which was filled with thin blood-stained fluid. This was sucked out with a syringe, and sent to the laboratory for examination; the fluid was about 10 c.c. in amount. A few masses of currant-jelly like medulla were also removed and sent to the laboratory, as was a section from the cyst wall. There were no areas of whitish tissue, characteristic of fibrous osteitis. The interior of the expanded cortex was curetted with Volkmann's sharp spoon, the opening in the cortex being enlarged until about three inches long and three-fourths of an inch broad, the defect extending from the level of the epiphyseal cartilage to the insertion of the deltoid muscle. At these limits the cortex and medulla seemed to become normal. The cortex on the median side of the bone opening was then crushed in (Fig. 91), and the wound was closed in layers without drainage: first the periosteum, then the deep fascia, and finally the skin. The time of the operation was one hour.

The Esmarch band and Wyeth's pins were removed after the wound had been dressed. A shoulder cap was applied, and the arm bandaged to the side.

At the first dressing, January 22, all the skin sutures were removed. There was moderate serous discharge (subcutaneous) from below the lowest suture. The rest of the wound was healed. The arm was dressed again three days later, when there was no discharge, the wound being soundly healed. The boy was sent home this day, two weeks after operation.

There have been no symptoms since operation, and at the end of six months, when reading proof, there is no evidence of recurrence. Fig. 93 is from a photograph made two months after operation, and Fig. 94 from a skiagraph made five months after operation.

PATHOLOGICAL REPORT. Dr. C. Y. White, Director of the Pathological Laboratories, sends the following report on the specimens submitted to him for study:

CYST WALL. Small fragments of the inner surface and wall of the cyst showed on histological examination normal bone trabe-

culæ, faced by a narrow margin of fibrous tissue. This latter separated the cyst contents from the bony structure and consisted of typical fibrous tissue cells without other cellular infiltration.

The bone fragments consisted of normal bone trabeculæ without cellular infiltration other than is common to spongy bone.

CYST CONTENTS. The contents of the cyst showed yellowish fluid containing blood clots. Smears made from the fluid showed the following cellular contents:

	Per cent.
Polymorphonuclear leukocytes	14
Mononuclear cells	2
Transitional cells	1
Lymphocytes	72
Eosinophiles	2
Neutrophilic myelocytes	9

No nucleated red cells; no giant cells; a few fibrin fibrillæ and serum.

Bacterial Cultures: Anaërobic and aërobic cultures negative. No bacteria were observed in the various smears examined.

Complement fixation test for syphilis made on the fluid from the cyst gave a negative reaction.



FIG. 94.—Skiagraph made May 29, 1913, nearly five months after operation, showing restoration of normal bone.

EYE DISEASES IN MANILA¹

By HENRY WINSOR, M.D.
SURGEON TO THE DISPENSARY OF THE HOSPITAL

DURING the years 1908 and 1909, while attending physician to the University Hospital, and St. Luke's Dispensary, Manila, P. I., it became my duty to report the work done in this dispensary, including that of the eye. In reviewing this report, I find that much of the work was performed by Major O'Connor, U. S. A. and Dr. Lankowsky, oculist to the Philippine General Hospital, U. S. A. Many of the cases here reported came under them as chiefs in charge, and all due acknowledgement must be made to their superior skill as specialists; for part of the time I was their assistant, at other times I had charge of the eye work myself.

Most of the patients were Filipinos; many, however, were Chinese. Race, climate, and social state have all had their influence on the character of the diseases mentioned in the summary appended, *e. g.*, retinitis pigmentosa was observed only in the Filipinos; trachoma especially in the Chinese. Sunlight had its influence as well as climate. There were more than 6000 new patients treated at St. Luke's Dispensary, 637 of whom were eye-patients.

¹ Read before the Episcopal Hospital Clinical Society, September 16 1912.

SUMMARY OF DISEASES TREATED

Amblyopia, toxic.	5
From nicotine (smoking)	4
From betel-nut (chewing)	1
Of the conjunctiva	138
Conjunctivitis, catarrhalis.	52
Conjunctivitis, phlyctenular	14
Conjunctivitis, trachoma	35
Pterygia	29
Pinguecula	3
Subconjunctival ecchymosis	5
Of the cornea	170
Abrasion	1
Abscess	3
Hypopyon, keratitis	3
Keratitis, simple	98
Leucoma.	16
Staphyloma	13
Ulcer	35
Wound	1
Of the eyeball	47
Bulbar atrophy	7
"Collapse of"	1
Foreign bodies on	9
Glaucoma	24
Panophthalmitis	2
Panophthalmitis from Graves' disease	1
Gonorrhoeal ophthalmia	3
Of the eyelid	53
Blepharitis, marginal	25
Blepharitis, ciliary	3
Ectropion	4
Furuncle	3
Cellulitis	2
Entropion	2
Hordeolum	4
Meibomian cysts	7
Sebaceous cysts	1
Symblepharon	1
Wounds	1

Of the iris	17
Iritis	9
Prolapse	1
Mydriasis	3
Synechia	4
Of the lachrymal apparatus	17
Dacryocystitis	16
Dacryops	1
Of the lens	92
Cataracts	91
Cataracts, traumatic	1
Of the nerves	38
Oculomotor paralysis and mydriasis	2
Oculomotor paralysis and ptosis	1
Oculomotor paralysis and nystagmus	1
Optic atrophy	26
Esophoria	2
Supra-orbital neuralgia	4
Supra-orbital neuritis and herpes corneæ from malaria, with hyaline bodies in R. B. C.	2
Of the retina	5
Retinitis hemorrhagic	1
Retinitis "pigmentosa"	3
Chorioretinitis	1
Of the sclera	2
Hyperpigmentation of sclerocorneal margin	1
Scleritis	1
Tumor of the eyelid	2
Refractive errors	51
Strabismus, internal	1
Asthenopia	7
Presbyopia	4
Myopia and hypermetropia	39

Of special interest are the toxic amblyopia patients; four suffered from dimness of vision due to nicotine poisoning. The Filipinos are incessant smokers, but do not chew tobacco, using betel-nut instead. The seeds of nuts of acacia catechu are rolled in the leaf, they are mixed with quick-lime, and chewed by the natives. Betel-nut (areca-nut), according to Gould's Dictionary is an astringent, tonic, stimulant, aphro-

disiac or erotic, and capable of increasing endurance. Arekane is a gum resin, a purgative prepared from betel-nut. The veterinarians use areca-nut combined with turpentine in capsules as a vermifuge. I have so used it with success for worms, both round and tape, in dogs. Nevertheless the Filipinos are frequently the subject of ascaris infestation in spite of their betel-nut chewing. One patient, a constant abuser of this habit, presented marked dimness of vision; his refractive error was slight (Dr. O'Connor) and did not require corrections. He presented no other symptoms, and it is the only instance of this disease that I have seen mentioned.

Another patient of Major O'Connor's excited interest. While operating for cataract, he observed that as soon as he touched the patient's eye, the latter was partially withdrawn into the socket. This withdrawal occupied about one or two seconds, the corneal and bulbar conjunctiva became apparently wrinkled, the eye presented the appearance common in the cadaver, in which death has taken place several hours previously. On removal of pressure the eye resumed its natural appearance in a few seconds, appearing gradually to fill up again. The Major made a successful enucleation of the cataract, with good result. I have never seen any mention of such a condition in the literature. The muscles are not supposed to have such actions, and the only explanation that I can afford is that this is a vasomotor phenomenon. Probably all of us have observed the eyes in exophthalmic goitre. Where the patient is excited the eyes appear more prominent than before, on removal of excitement the eyes return to their habitual position. According to Möbius, who has made a most careful study of goitre, the exophthalmos is a vasomotor phenomenon. The vessels relax, fill with blood, exude serum into the eye, the latter enlarges especially forward in the direction of least resistance. While there were no symptoms of Graves' disease in Dr. O'Connor's patient, I consider the vasomotors to have been responsible for this very unusual action on the part of the eye. Had

it been muscular, the withdrawal would have been more suddenly performed, the return to normal quicker, wrinkling of the coats of the eye could scarcely have taken place. I am unable to find any evidence of such actions by the intra-ocular muscles, and last of all, were such an action possible, it would have acted bilaterally, so that both eyes would have been withdrawn, which was not the case. According to all authorities, the eye muscles coördinate bilaterally.

There were three gonorrhœal ophthalmia patients treated by Dr. Lankowsky, who on questioning them discovered that they had put their own urine into their eyes, with the hope of curing trachoma, from which they suffered. I demonstrated intracellular diplococci in the ocular and urethral pus of all three patients, at Dr. Lankowsky's request. He cured the ophthalmia with silver nitrate solutions, the trachoma disappearing at the same time. While Dr. Lankowsky always censured the patients for these attempts at self-cure, nevertheless in intractable trachoma, under suitable regulations in a hospital, some modification of this method of cure has been tried elsewhere with success. There are said to be two kinds of trachoma in Manila. There were 35 trachoma patients treated in St. Luke's Dispensary.

In 49 cases of corneal ulcer, I demonstrated hyaline bodies in the red blood cells of two. There were probably other examples of malarial keratitis but they escaped my observations and study. No other cause for ulcer was discovered in the two patients above mentioned.

Captain O'Connor showed me two patients with retinitis pigmentosa, and I afterward had two such patients of my own. All exhibited night blindness, and were apparently beginning to lose their sight by day. None had a true retinitis. Pigmentary deposit was moderate and scattered in small masses. I consider these cases together with the one of hyperpigmentation of the sclerocorneal margin, which Dr. O'Connor so kindly showed me, to be due largely to an over-zealous effort on the part of nature to accommodate itself

to the tropical glare of the sun, this being excessive on account of the direct light and the neighborhood to the sea. This disease is apparently commoner among the dark-skinned races, who also exhibit excessive pigmentation elsewhere in the body, notably on mucous membranes, *e. g.*, the bladder mucosa may be so dark as to make cystoscopy difficult, as in negroes and Filipinos, while the hard palate, gums, and lips are frequently the subject of excessive pigmentary deposits. The disease is commoner in the swarthy, rarer in blondes, while it probably never exists in albinos, where pigment is almost absent. White inhabitants of tropical countries become darker, and may show variations in pigmentary deposits on the hands. Descendants of white races become darker than their ancestors.

One patient consulted me for an optic neuritis of one eye. I referred her to Dr. Lankowsky who informed me of the true nature of the case. The central artery of the retina had become blocked by an embolus, and the Doctor suggested an uterine origin. I made a vaginal examination and found the uterus, the size of one's fist, with a foul cervical discharge. The patient then gave a history of an abortion three months previously.

There were 57 eye operations performed in the hospital; 49 of which were performed by Drs. O'Connor and Lankowsky. All were cured or improved, including 28 cataract extractions, 5 enucleations of eyeball, 7 iridectomies, 2 for pterygia, 2 for pupillary membrane, 1 strabotomy, 3 tenotomies, and 2 for tumors of eyelid. I performed only six cataract extractions, one being bilateral, one enucleation of eyeball for panophthalmitis, and one operation for eversion of the lower lid, all with improvement.

There were 2525 visits made by patients to the eye dispensary, 637 new eye patients among 6000 new patients treated.

I wish to acknowledge, once more, my indebtedness to Major O'Connor and Dr. Lankowsky for their kind instructions in this work during the time I was their assistant.

PSEUDOGLIOMA IN CHILDREN¹

By FREDERICK KRAUSS, M.D.
OPHTHALMOLOGIST TO THE HOSPITAL

E. M., aged eight years, presented himself at St. Christopher's Hospital for children, January, 1912, having been referred by the school physician on account of defective vision. A peculiar greenish yellow reflex from the widely dilated pupil was immediately noticed, the appearance of the so-called "Katzenauge." The eye was blind.

Family History. Negative; father, mother, two sisters, and one brother living and well.

Personal History. Had measles, whooping cough, and chicken-pox. General health always good. The patient is a well-developed and healthy boy of eight years.

The mother states that the labor at birth of the child, though normal, had resulted in an injury to the left eye, the child being unable to open the eye for three or four days. The eye was normal in every way, until seven months ago, when there was a slight redness and inflammation of the left eye, associated with some photophobia, but no pain. Several attacks of similar nature had occurred since then. The mother had not noticed any change in the boy's vision, and paid no attention to his eye until last September, when she noticed a peculiar appearance in the temporal side of the left pupil, but did nothing until her attention was called to it by the school physician.

The vision in the right eye is normal, the left eye blind in the strongest light stimulus.

In the right eye the pupil responded to light accommodation and convergence. The media were clear and the eye ground normal. The muscular movements of both eyes were normal.

¹ Read before the Section of Ophthalmology of the College of Physicians of Philadelphia, January 16, 1913

In the left eye the cornea was clear, the anterior chamber normal in depth, except on the temporal side where it was shallow, due to some bulging of the iris. The pupil was immobile and three-fourths dilated. There were a few narrow synechia between the lens and iris on the temporal side, with a few spots of pigment on the anterior capsule of the lens.

By reflection of light from the window, a greenish-yellow reflex was apparent, due to what appeared to be a newgrowth on the temporal side, directly behind the lens. With the ophthalmoscope, there was a dustlike haze of the vitreous, permitting a red reflex from the nasal half of the retina, but no details. The tension was normal or perhaps slightly plus. One week later, the tension suddenly diminished to minus two. The greenish-yellow reflex gradually extended all about the pupil. The vitreous cleared up slightly, so that the growth which was immovable, was seen to be close behind the lens on the temporal side, slightly deeper on the sides, and of greatest depth in the centre and to the nasal side. About six weeks later the vitreous cleared so that the vessels could be seen on the surface of the tumor. This was especially marked to the nasal side of the centre where there was an appearance resembling that of the optic disk, with an irregular but well defined outline, with vessels extending therefrom, best seen with plus 16 D.

A diagnosis was based upon the evidences of an old iritis, vitreous opacities, and a low tension, all indicating a pseudoglioma, especially since there was no further increase of tension during a period of observation of several months in my clinics at St. Christopher's Hospital for Children and the Protestant Episcopal Hospital. About May, 1, there was a sudden rise in tension, to slightly above normal, which aroused some doubt as to the absolute accuracy of the diagnosis of pseudoglioma, and I insisted upon running no further chances. The parents consented and the eye was enucleated, May 4, 1912, at the Episcopal Hospital. The specimen was placed in formalin solution and sent to Dr. C. Y. White, pathologist to the hospital. There was normal healing with good socket.

This is the case of growth extending into the vitreous, which I presented before this section last spring. It was then diagnosticated as pseudoglioma by the majority of Fellows present, but as there was some doubt it was the unanimous opinion that the eye should be enucleated.

Dr. C. Y. White, Director of the Pathological Laboratories of the Protestant Episcopal Hospital of Philadelphia, reported as follows:

Preparation of Specimen. Formalin 10 per cent. Sectioned in half. One-half mounted in celloidin for histological study, the other half prepared for permanent mount in gelatin.

A delicate whitish cone-shaped hollow (wilted morning glory) membrane stretched from the region posterior to the lens to the optic nerve. The apex of the cone was attached to the nerve while the base was irregularly fluted and folded inward just back of the lens, with slight attachment in the ora serrata region. In the posterior half of the eyeball it forms the solid stem of the morning glory.

Microscopic Examination. The above membranous formation was seen to be composed of detached retina which had separated from the pigment layer of the chorioid. The detachment was complete with the exception of the nerve and anteriorly in the region of the ora serrata.

Between the detached retina and the pigment layer of the chorioid there is a subretinal exudate which completely filled this cavity, but which shows shrunken in the histological specimens.

There is a large subretinal hemorrhage in the nasal side of the ora serrata and several smaller hemorrhages both recent and old in the detached retinal structure. An inflammatory exudate partly organized lies posterior to the lens surface (most marked on the outer side) and between the lens and the folded base of the detached retina. This exudate shows evidence of blood pigment from a former hemorrhage and an increase in the fibrous tissue, the result of organization.

The retina shows dilated and engorged bloodvessels with thickened walls. The opposed surfaces of the detached retina do not show adhesions with the exception of the region of the nerve where the surfaces are so close together it seems impossible to separate them by the microscope. The normal structure of the retina seems to be distinctly degenerated in areas, evidenced by poor staining and cellular infiltration present. The chorioid and sclera were attached throughout and were apparently normal.

Microscopic Diagnosis. Detached and degenerated retina with subretinal hemorrhages and exudate.

The difficulty in making a diagnosis between glioma and pseudoglioma occurring in children is demonstrated in this

case. Even macroscopic examination of the section is strongly suggestive of tumor, the microscopic examination being necessary to exclude glioma retinae. We are further confronted with the distinction between so-called pseudoglioma, by which is usually meant a form of suppurative chorioiditis, and retinitis proliferans.

Hyperplastic hemorrhagic retinitis, or as it is more commonly called retinitis proliferans, is an exceedingly rare and interesting disease. Schobl has seen but five cases in a series of 179,057 patients. Weeks has observed two cases in a series of 24,000 private cases.

Its etiology is obscure but it is generally recognized to be due to the organization of a blood clot in the vitreous. Ordinarily in hemorrhage into the vitreous the blood clot is almost completely absorbed as we see in the recurrent vitreous hemorrhages in adolescence. This is probably due, according to Parsons, to the absence of fibroblasts in the vitreous and their scantiness in the retina, for the retinal connective tissue consists of neuroglia and epiblastic structure which probably takes no part in fibrous tissue formation. The only mesoblastic tissue in the retina is that forming and surrounding the retinal bloodvessels.

In some cases, however, blood clots may organize, giving rise to masses of fibrous tissue in the vitreous, vascularized by new-formed bloodvessels derived from the retinal system. This condition is known as retinitis proliferans. This tissue is most commonly situated near the disk, and the vessels spring from this neighborhood, probably owing to the fact that there is more mesoblastic tissue here than in other parts of the fundus.

He states further that there seems to be some special factor necessary to stimulate organization and this is found in some general diathesis. In nearly all these cases there is either a history of syphilis or the patient is suffering from nephritis, diabetes, or some other toxæmia.

Weeks defines retinitis proliferans as an affection charac-

terized by the appearance of white bands or membrane of newly formed connective tissue which springs from the retina and extends into the vitreous, being accompanied in many cases by new-formed bloodvessels.

Collins and Mayou state that the continuity of the hyaloid membrane of the vitreous plays a similar part to that of the membrane of Bruch, in preventing the spread of endothelial cells from the retinal vessels to the vitreous. Leukocytes and toxins can, however, pass through it, the former constituting the vitreous opacities usually accompanying retinitis. When a rupture in the continuity of the hyaloid membrane occurs, which is usually the result of hemorrhage into the vitreous, endothelial cells may spread into it, causing the formation of bands of fibrous tissue, a condition known as retinitis proliferans. The subsequent contraction of these bands, often causes detachment of the retina from its pigment layer.

Etymologically considered, pseudoglioma means false glioma, or anything that has the surface appearance of glioma, but which is finally proved to be something else. Most authors regard pseudoglioma as an intermediate stage in suppurative chorioiditis. Collins and Mayou state that in suppurative iridocyclitis, although pus is formed in the vitreous, the inflammation may subside, with the result that organization in the exudate takes place and a fibrous membrane (cyclitic membrane) forms behind the lens giving rise to a yellowish reflex, a condition that may clinically resemble glioma of the retina, and hence has received the name of pseudoglioma. In pseudoglioma the retina becomes matted up with the exudate and the ciliary body to form a large mass in which the pigment cells proliferate and migrate. The chorioid, ciliary body, and ciliary process are pulled inward, opening up the suprachorioidal lymph spaces and the root of the iris backward into the circumlental spaces, deepening the periphery of the anterior chamber. The epithelium covering the inner surface of the ciliary body often extends into the

cyclitic membrane in the form of pigmented and partially tubular processes.

The following from Swanzy is very interesting. He states that "purulent chorioiditis does not reach the latter stage in every case, but may remain confined chiefly to the chorioid, vitreous humor, and iris. The pain in these cases is not severe, and the affection occurring in children may be mistaken for glioma of the retina, indeed the name of pseudoglioma has been given to it. It must be stated that very recent investigations go to show that pseudoglioma has its origin in the retina rather than in the chorioid. It is distinguished from the glioma of the retina by the muddy vitreous usually present with it, by the posterior synechia, and by the retraction of the pupillary part." It would seem from this description that Swanzy appears to recognize some difference between purulent chorioiditis and pseudoglioma in children, which we believe is amply justified.

Vossius states that Treacher Collins found that in 7 cases out of 24 eyes enucleated for glioma the diagnosis was incorrect; Vetsch and Isler, 4 times in 41 cases; Haab, 5 times in 20 cases; and Greef, 4 times in 21 cases. In nearly every case reported by Greef there was a retinal detachment caused by a lemon-yellow fluid in which there were many cholesterin crystals or fat cells with pigment crystals. He believes that the cause of the detachment was a chorioiditis following infectious diseases.

It is evident from the foregoing descriptions that our case of newgrowth belongs to neither section. Though there is evidence of hemorrhagic extravasation, the hemorrhage is subretinal and in the retinal tissue. There was no evidence of vitreous hemorrhage and there are no fibrous bands in the vitreous that could have detached the retina. The exudate is not in the region of the papilla but is subretinal, and almost exclusively in the region of the ora serrata. Retinitis proliferans is apparently excluded. Nor does it respond to the second description, namely, pseudoglioma as it is generally

described in the text-books. There is no disorganization nor marked inflammatory phenomena present in the iris, ciliary body, or chorioid. In fact they are practically normal. In suppurative chorioiditis the iris and ciliary processes are attached to a cyclitic membrane, pulling inward and opening the suprachorioidal lymph spaces. This pathologic appearance is absent in our case. The probable explanation of our case is as follows:

There is a history of trauma at birth, possibly causing trophic changes in the region of the ciliary body. At a later date, for some reason, subretinal hemorrhage occurs in this region with gradual separation of the retina from the chorioid by an exudation of blood serum. The repeated hemorrhages cause a slight iritis with transient inflammatory symptoms. The blood pigment is gradually absorbed, the smaller cells remaining to become more or less organized. Bulging of the iris was probably due to a hemorrhage anterior to the organized clot, pushing the lens forward. The gradual separation of the retina allowed degeneration in certain areas with hemorrhage into the retinal substance. There was no apparent hemorrhage into the vitreous.

The sudden rise in tension from minus two to slightly above normal was probably due to the large subretinal hemorrhage seen on the nasal side near the ciliary body, causing temporary occlusion of the canal of Schlemm.

It seems evident, therefore, that in those cases of pseudoglioma in which iridocyclitis can be excluded by history, we have a distinct disease originating between the layers of the retina and chorioid, resulting in complete detachment of the retina, with degeneration of the bloodvessels, hemorrhage, and the formation of a large exudate in the anterior portion of the eye which has a tendency to form a fibrous tissue, the whole appearance suggesting glioma of the retina. The observation of Greef that the subretinal liquid is of a lemon-yellow color in these cases indicates the reason for its deceptive appearance. The retinal vessels when not hidden by vitreous

opacities or exudate are sometimes visible, adding to the difficulty of diagnosis.

Dr. W. T. Shoemaker and C. M. Hosmer report a case of retinitis, proliferating in character, in a detached retina. The eye had been enucleated from a girl, aged twelve years, on account of a diagnosis of glioma of the retina. The findings were briefly a complete detachment of the retina, hemorrhage, formative tissue, new bloodvessels, leukocytes, and excess of supporting tissue, all resulting in the formation of a solid mass in the anterior vitreous, clinically resembling glioma. Absence of uveitis and of structural changes in the anterior segment of the eye. There was a history of trauma seven years before. The changes in the chorioid were limited to a small area of chorioiditis, which, owing to its minuteness and position near the ora serrata, was considered of slight importance. The process seemed to be limited to the retina.

Treacher Collins is quoted as thinking that this type of pseudoglioma commences as a retinitis, though later stages pointed rather to primary cyclitis and retinitis. In one case, however, the condition remained retinal for at least three years.

Eversheim reports three cases in which the eyeball was removed on account of glioma retinae:

The first eye examined was from a child, aged twelve years. The retina was completely detached, showing areas of degeneration. The chorioid was nearly normal. There was marked exudate behind the lens with evidences of hemorrhage. The subretinal space was filled with a homogeneous fluid containing numerous cholesterin crystals. There was no evidence of rupture of the retina. The retinal bloodvessels showed marked degeneration with thrombosis and hemorrhage.

In the second case the right eye was enucleated from a child, aged four and one-half years, on account of glioma retinae. The retina was completely detached with the chorioid adherent, the whole mass contracted to a space of 5 mm. behind

the lens. The retina was degenerated, with a mass of exudate between the chorioid and retina anteriorly. There were marked pathologic changes in the bloodvessels, with evidence of hemorrhage and thrombosis markedly similar to the first case. The subretinal fluid was the same.

In the third case from a child, aged two and three-fourths years, in which there was the appearance of amaurotic "Katzenauge" with vessels appearing on the surface of the growth, the eye was enucleated on account of a diagnosis of glioma retinae. The retina was completely detached from the ora serrata, the retina folded forward, completely covering the ciliary body, zonula, and posterior surface of the lens. In the centre of the latter, both parts of the retina meet and pass backward close together toward the papilla. The same retinal and vascular degeneration was present as in previous cases, with remnants of hemorrhage and exudative processes.

The greatest change in the bloodvessels and the largest hemorrhages were close to the ora serrata. The chorioid with its pigment layer was adherent to the sclera, and showed but little change.

The microscopic findings led Eversheim to the following conclusions:

The disease begins primarily or metastatically at some place on the retina, probably in one of the central vessels. Connected with this there is a high-grade nutritional disturbance, or retinal hemorrhage, causing retinal degeneration. The hemorrhages take place between the retina and chorioid and in the retinal substance. The cellular infiltrate can be traced to excitation produced by the hemorrhage as suggested by Von Hippel. Fibrous tissues may be present through organization of the hemorrhage. When retinal detachment occurs, the hollow spaces are filled with hemorrhage and exudate from the diseased bloodvessels. This exudate may be so extensive as to push the lens forward with closure of the angle of the iris with rise of tension, giving symptoms of glaucoma and making diagnosis very difficult. These findings he believes

seem to indicate that pseudoglioma in a majority of cases is based upon definite pathological basis and is a disease of the retina and not of the chorioid.

I regret that on account of lack of time I have been unable to make a thorough search of the literature for further similar cases, but hope to do so at some future date.

In conclusion, I will repeat that there is some evidence to believe that there is a distinct disease of the retina occurring in children which produces clinical symptoms closely simulating glioma of the retina. This disease is most properly termed pseudoglioma since its appearance is such that the most able diagnosticians cannot make a sure diagnosis previous to enucleation and microscopic study.

The examination of all these cases indicates that the retinal detachment is caused by an outpouring of a yellowish fluid between the pigment layer and retina which gradually involves the whole retinal tissue. Hemorrhages and exudate seem to be confined to the anterior part of the eyeball, and are generally in the subretinal space and in the substance of the retina.

Finally these cases are not associated with suppurative chorioiditis and the term of pseudoglioma should be divorced from its connection with that disease. The history of the previous intense inflammation is a safeguard from a false diagnosis of glioma.

FIG. 95.—Fundus, right eye, boy, pupil dilated. Showing absence of choroid at the periphery; more complete space formation, with heaping of pigment at the borders; ciliary vessels exposed; posterior staphyloma; fine granular pigmentation.

FIG. 96.—Fundus, right eye, girl, pupil dilated. Showing absence of choroid at the periphery; attempt at space formation with heaping of pigment at the borders; ciliary vessels exposed; fine granular pigmentation.



CHOROIDEREMIA, REPORT OF TWO CASES IN ONE FAMILY¹

By HAROLD G. GOLDBERG, M.D.

ASSISTANT TO THE OPHTHALMOLOGIST OF THE HOSPITAL

THERE was nothing of interest in the parental history of the two cases; the children were respectively twelve and twenty years of age. They were of a family of four, the sexes equally divided. The mother is living, and in good health; the father died of renal calculus.

The condition of the girl, aged twelve years, the first to be described, was unknown, in fact unsuspected by any member of the family, and was first discovered by the school medical inspector who advised the mother to have the child refracted for a visual error which fell below the requirements for the school test. They first consulted a refracting optician, whose correction was worn for about three months, comfortably, it appears, and the only reason for applying at the hospital was upon the advice of the school physician. At the time of our examination it was found that the vision in the right eye equalled $\frac{5}{22}$, and in the left $\frac{5}{60}$; her correction did not improve this. The external appearances of the eye were normal, the media were clear, but the following unusual fundus condition was discovered. Including the entire periphery of the fundus, to a point well within the view of the ophthalmoscope, with the pupil undilated, there was found a complete absence of the choroid. The sclera was entirely exposed, was yellowish-white in color, and was abundantly traversed by bloodvessels, both retinal and ciliary. This area was separated from the normal choroid by an irregular line of pigmentation,

¹ Read before the Episcopal Hospital Clinical Society, November 18, 1912.

something like that seen after extensive atrophy, with the heaping of the pigment at the borders. This was the first impression one received, but upon more careful study it was found that the line was more cellular in appearance, as though it were composed of an arrangement of healthy pigment cells, and not the detritus of degeneration we find in diseased processes. The line of demarcation presented the appearance of a constant attempt at space formation, which was rather deceptive; in some positions the spaces were well formed, in most, however, they were incomplete. Elsewhere, the choroid was normal in appearance, there were occasional patches of the same character of pigment, without any definite arrangement, scattered throughout the fundus; while an incomplete choroidal ring on the nasal side was also similar. The nerve and retina were normal, the macula was slightly granular.

In the case of the boy the condition was much more marked. His history is as follows: aged twenty years, is poorly developed mentally, and appears much younger in every way. From his early childhood, it has been known to his family that his vision was defective, but very little attention has been paid to it. He has never worn anything but an optician's correction. He attaches no importance to his poor vision, although it has disqualified him from almost every kind of work, and a well marked night blindness has added to his difficulties. His vision in each eye equals $\frac{5}{60}$. His cornea was clear, and the iris normal. With the ophthalmoscope it was found that he had in each eye a stellate nuclear opacity of the lens. The vitreous was normal, and the fundus presented the following appearance: The choroid was represented by an irregular ring, varying in breadth by ophthalmoscope measurement from 4 to 6 centimetres; its narrowest portion was below and to the nasal side. It was encroached upon, both from the aspect of the nerve, and from the periphery. It was less healthy in appearance than in the first case described, but its general characteristics were the same. The periphery, with the exception of an occasional patch of pigment, was entirely bare, a little paler yellow, and was traversed in the same manner, by the same character of bloodvessels. There was more complete space formation, the irregular line of pigment was less dense, so that its general character was more easily determined. The appearance of this pigment gave even more the impression of healthy cells, and not detritus, and suggested the possibility that this was actually the retinal pigment epithelium. That portion of the choroid which was intact, was apparently normal, presented the same character-

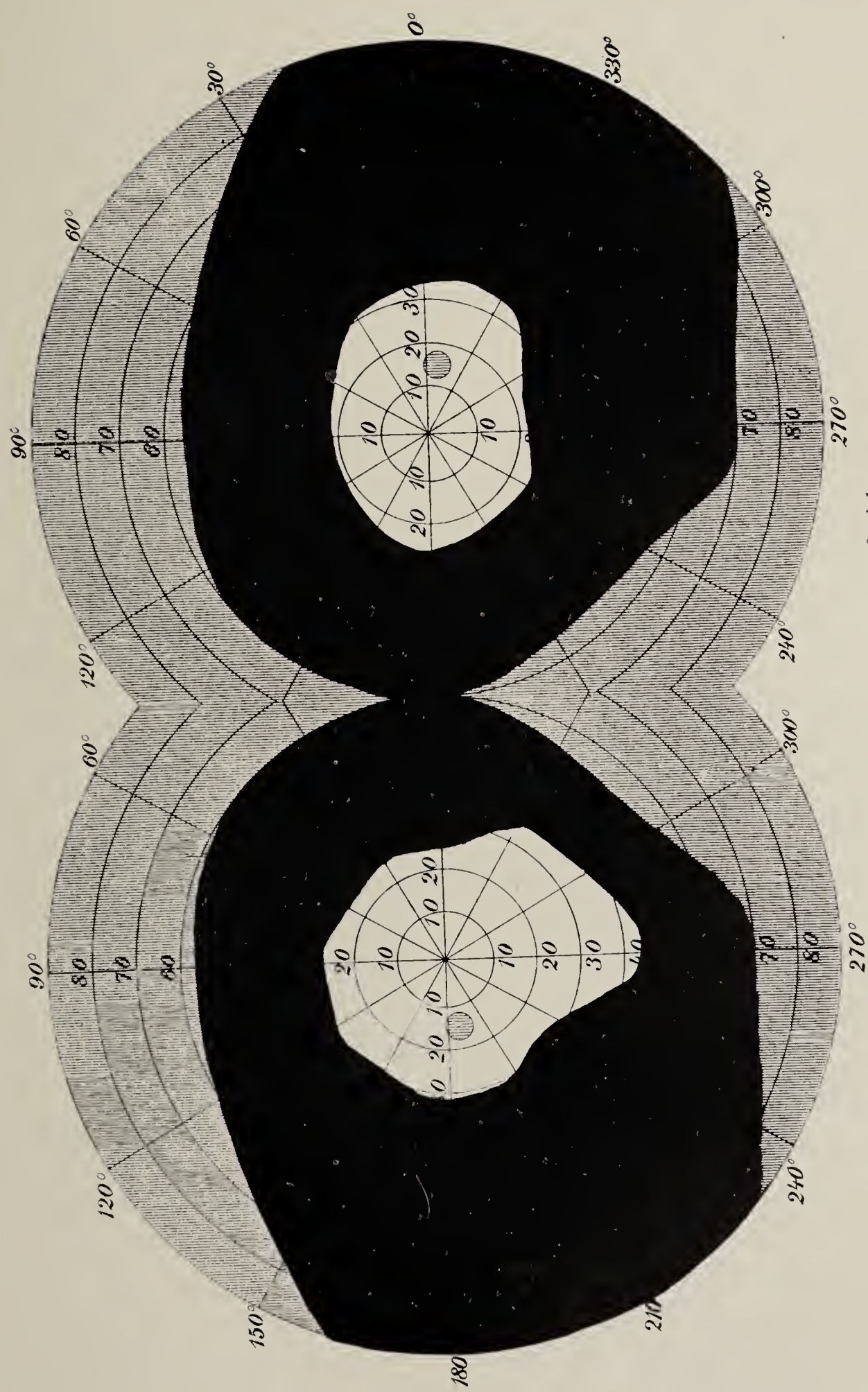


FIG. 97.—Visual field chart of girl.

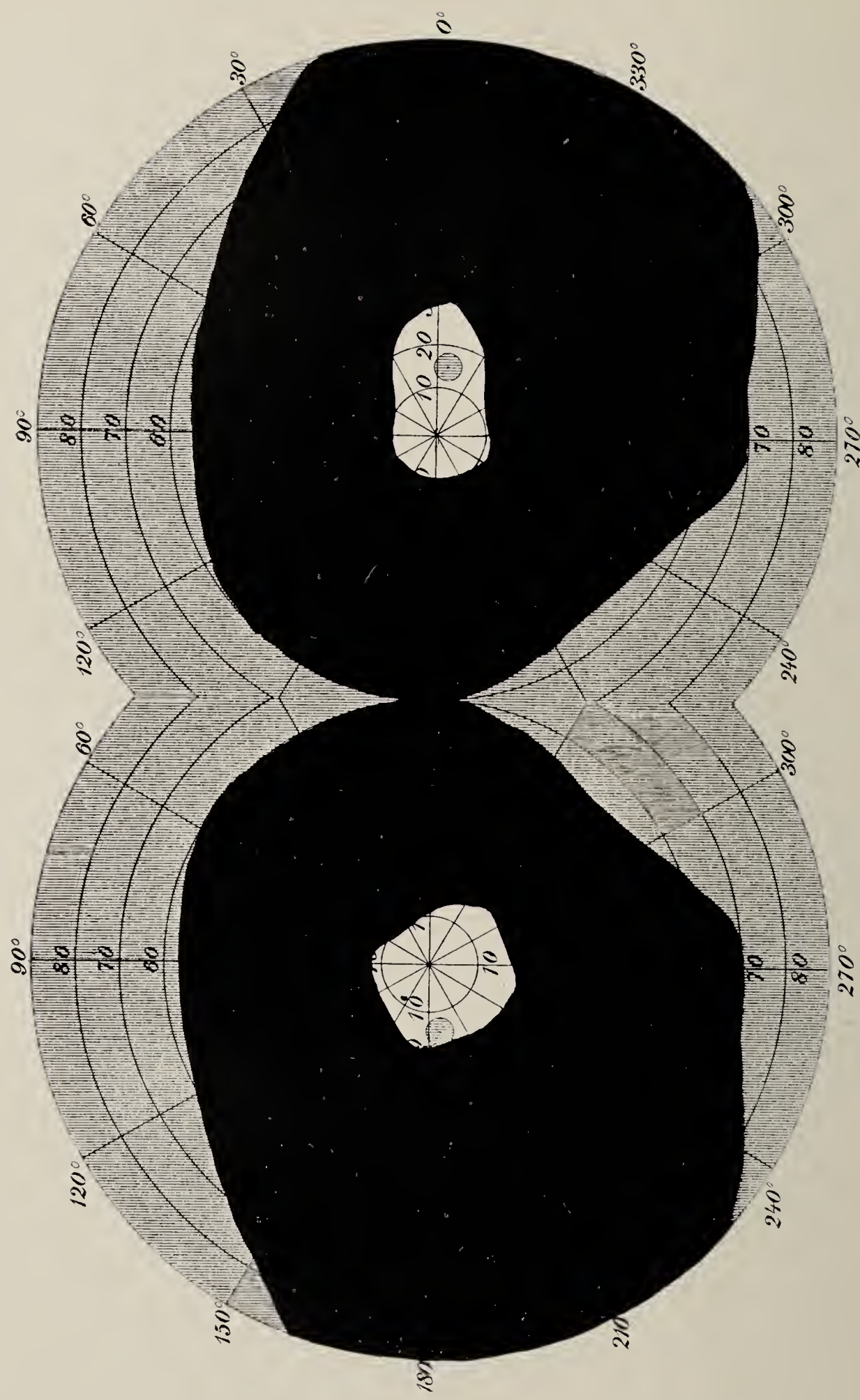


FIG. 98.—Color fields of girl.

istics as in the first case, with the same isolated patches of pigment, which in this case resembled ink splashes. The absence of the membrane at the disk may have been due to a staphyloma; this was not out of the question, by reason of the general weakening of all the coats of the eye. It was impossible to determine definitely, however, because of the high-grade of refractive error, and the lenticular opacities which partially obscured the ophthalmoscopic view.

The girl's refraction with a mydriatic equalled: right eye, —S. 1.50 D. \bigcirc + C. 2.50 D. ax. $100^\circ = \frac{5}{15}$; the left eye, —S. 2.00 D. \bigcirc + C. 4.00 D. ax. $80^\circ = \frac{5}{15}$. The refractive error of the boy could not be measured with sufficient accuracy to make a correction of any value to him. He also had a mixed astigmatism, of higher degree than the girl.

The fields in each case, for both form and color, were found to be very much contracted, and in neither case could the physiological blind spot be demonstrated. In the girl, this may have been due to a certain lack of intelligence, but in the boy it was probably entirely absent.

These two cases, represent, I believe, that rare form of congenital anomaly known as choroideremia. The condition is generally spoken of as "probably congenital;" so far as I have been able to learn, no two cases from the same family have ever been described. This, in itself, is very strong in support of the belief in the congenital origin of the condition. But, in addition, in the case of the boy, we have the lenticular changes, and general mental characteristics, sometimes found in this group.

It seems to me, the better theory to account for the anomaly is that described by Collins and Mayou as a failure in development due to a failure in the formation of bloodvessels in the inner part of the mesoblast which surrounds the secondary optic vesicle. The region of the cleft in the secondary optic vesicle, through which the mesoblast grows up to form the vitreous, and where for a time there is a connection between the intruding mesoblast and the encircling mesoblast, is the position in which this failure in vascularization most frequently occurs, giving rise to typical coloboma. A delayed

separation of the intruding mesoblast from the encircling mesoblast is the probable cause of the defective vascular formation. The choroid receives its chief blood supply from the short ciliary arteries. These are about twenty in number and perforate the sclerotic in the vicinity of the optic nerve. They proceed from two main trunks which arise from the ophthalmic artery. A failure in the formation of these vessels, except those which supply the region of the macula, would account for the condition described in my two cases. The ciliary body and iris which receive their blood supply from the long posterior ciliary arteries and the anterior ciliary arteries are not affected. The large ciliary vessels which are probably seen out near the periphery are most likely branches of the long posterior ciliary arteries, which perforate the posterior part of the sclerotic further forward than the short ciliaries, and pass to the ciliary body before breaking up. Some of them may also be veins proceeding from the ciliary body to the vortex veins which perforate the sclerotic at the equator.

These two cases occurred in the service of Dr. Bromley, through whose kindness and courtesy I am permitted to report them. I beg to acknowledge this privilege with thanks.

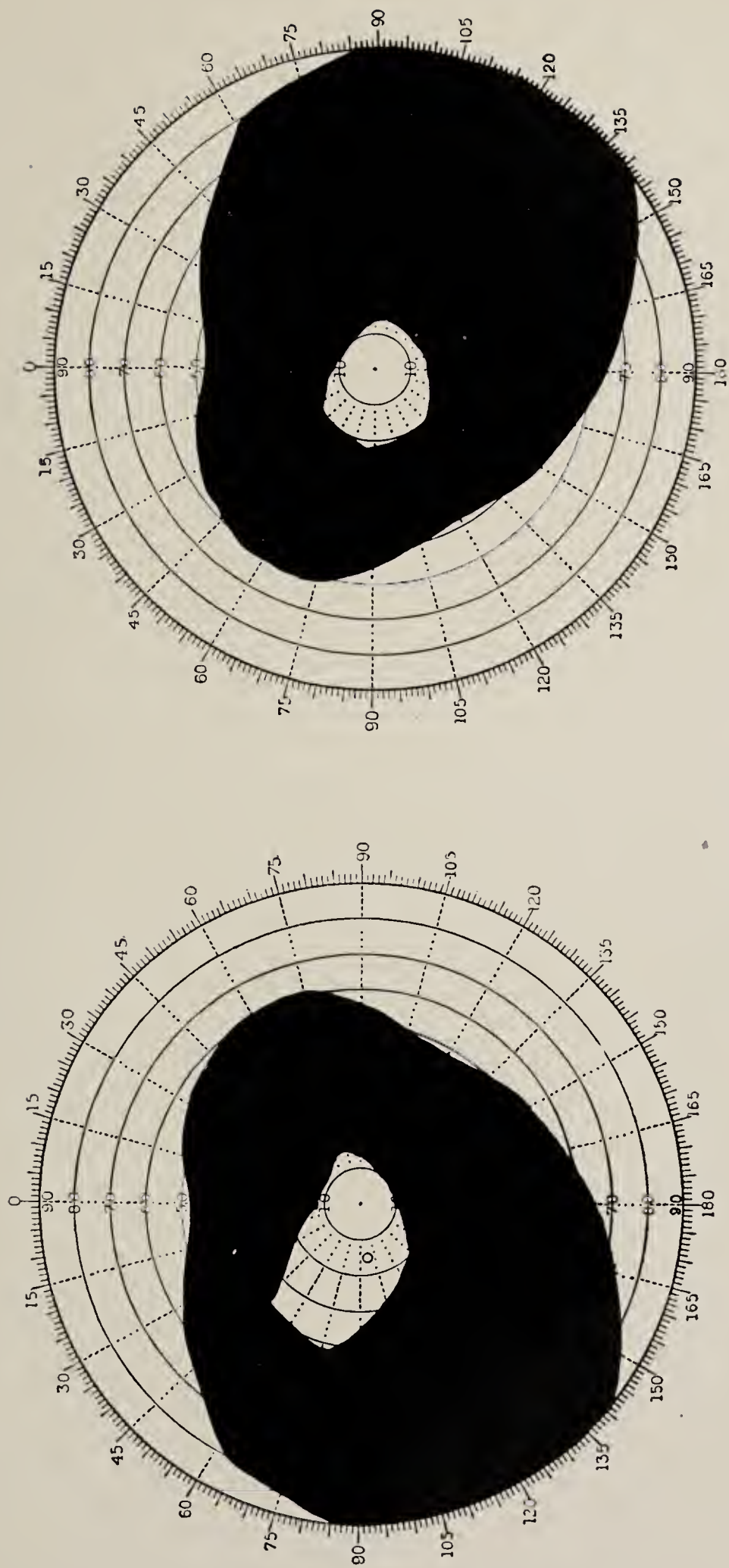


FIG. 99.—Visual field chart of boy.

SOME ORAL CONDITIONS MET WITH IN HOSPITAL PRACTICE¹

BY ROBERT H. IVY, M.D., D.D.S.
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My purpose is to call to your attention some of the lesions of the teeth and jaws that are commonly seen in hospital patients, and the recognition of which is often an important factor in the treatment of the disease for which the patient was admitted.

The importance of the mouth as an avenue for infection of the body is now well recognized. William Hunter says, "My clinical experience teaches me that if oral sepsis could be excluded, the other channels by which medical sepsis gains entrance to the body might almost be ignored." Practically all infection of the body, barring injury and venereal disease, *must* gain entrance in the first place by way of the oral cavity.

I will first outline some of the commoner lesions of the teeth and gums, the pathology of which is only imperfectly understood by the medical profession as a rule. The entire sequence of dental disease, from beginning decay of the enamel to alveolar abscess, together with other lesions such as tartar and pyorrhea alveolaris, is usually grouped under the comprehensive term "dental caries," without any attempt to distinguish one stage from another. An instance of the ignorance of oral pathology even among the highest surgical authorities is seen in a recent system of surgery, where the

¹ Read before the Episcopal Hospital Clinical Society, November 18, 1912.

contributor describes alveolar abscess as "due to a carious spot about the fang of a tooth." In examining a large book, one naturally turns to the subjects in which he is especially interested, and it is hoped that this is not a sample of the general standard of the work.

Dental caries, or decay of the teeth, is brought about by a solution of the inorganic salts of the enamel and dentine by acids, which are produced by the action of bacteria on carbohydrate food. The result is a cavity in the tooth. Any further conditions are sequelæ or results of dental caries. The resident physician is often called to see a patient in the ward suffering from toothache, and having made the diagnosis of toothache, regardless of the stage of dental disease present, prescribes oil of cloves, which is applied as a rule by the nurse somewhere in the region where the pain is felt. After a while the tooth may become tired of aching, and allow the patient to go to sleep. In the hope of assisting the resident physician to afford scientific relief from these conditions, I shall briefly outline the clinical course of dental disease and indicate the treatment of the different stages.

Clinically, the course of disease in a tooth may be divided into four stages, each presenting definite symptoms, and requiring special treatment: (1) Caries, without exposure of the pulp; (2) exposure and inflammation of the pulp; (3) death of the pulp and infection of the peridental membrane and (4) dento-alveolar abscess.

First Stage. The process of decay first destroys a portion of the enamel and then the dentine, forming a cavity which does not extend deeply enough to involve the pulp chamber. Pain may be entirely absent, and seldom calls for sedative measures. The chief importance of this stage to the physician is not to mistake it for discoloration of the teeth by calcific or other deposits in weighing in a given case the effect of the oral condition on the general health of the individual. A simple exploration of the tooth with a fine probe will usually determine whether a dark area is due to a cavity.

Second Stage. The carious process has uncovered the sensitive pulp of the tooth, setting up dental myelitis or pulpitis. Here we may have severe pain on slight provocation by trauma in chewing food, or from contact with hot or cold food, fluid, or air. The pain is of a sharp throbbing character, made worse by lying down. The tooth is extremely sensitive to a spray of cold water, but not very tender to external pressure. Examination shows a cavity, in which it is sometimes possible to see the bright red inflamed pulp, though this is usually covered by a layer of diseased dentine and *débris*.

Third Stage. The increased blood supply to the inflamed dental pulp causes engorgement, and finally, strangulation and gangrene. All symptoms may disappear for a varying length of time. Invasion of the necrotic pulp tissue by putrefactive bacteria next takes place, gas forms, and the infection is forced out through the apical foramen of the root into the surrounding peridental membrane. The symptoms that now arise are not those of inflammation of the live pulp, but of the peridental membrane. The pulp is dead, and therefore the tooth is not sensitive to cold, nor does passage of an instrument into the cavity cause pain. The pain that is present is of a dull boring character, and the tooth appears to be elongated, that is, it is pushed slightly out of its socket from swelling of the peridental membrane, and it strikes teeth of the opposing jaw before its fellows when the mouth is closed.

Inflammation and death of the dental pulp may take place under an artificial crown or filling, or in connection with an apparently healthy tooth where there is a history of trauma.

Fourth Stage. If the infection of the pericementum be not successfully overcome, pus forms, and we have an acute dento-alveolar abscess. In addition to the symptoms of pericementitis, a swelling appears in the tissue about the tooth, and may be so great as to be visible externally. Sometimes this swelling can be detected only by palpation with the finger in the vestibule of the mouth over the roots of the teeth. Fluctuation is usually obtainable. The formation

of the abscess is usually accompanied by a rise in temperature. The abscess may point rapidly and break spontaneously, usually in the vestibule of the mouth on the outer side of the alveolar ridge. Rupture of abscesses on the palatal surface or into the floor of the mouth is rare. Discharge of the pus usually gives instant relief from pain. If no further treatment be given, a painless chronic discharging sinus usually remains.

TREATMENT. The *first stage*, where we have a simple cavity in the tooth, usually does not call for treatment in the hospital.

Second Stage. Acute inflammation of a live pulp. Where there is a large cavity, the soft debris should be removed as thoroughly as possible with cotton soaked in hydrogen peroxide on a probe, in order to give any drugs applied a better chance to act directly on the exposed pulp. The most convenient application consists of a paste of carbolic acid and morphine, these ingredients being at hand in every hospital ward. A drop of carbolic acid is placed on a glass slab (an inverted medicine glass answers the purpose), and a one-fourth grain tablet of morphine sulphate incorporated with it. This paste is taken up by a small pellet of cotton half the size of the tooth cavity, the cavity is dried, and the pellet placed as nearly in contact with the exposed pulp as possible. A second pellet of plain cotton covers the first. A hypodermic injection of morphine may be given if necessary. Where the pulp is exposed and inflamed beneath a filling which is frequently loose or badly broken down, the filling can sometimes be removed without much difficulty, and the sedative application made. If not, the physician must rely on hypodermic medication until a dentist can be called to remove the filling, or else the tooth must be extracted. No amount of local applications outside the tooth will relieve the pain of this stage.

Third Stage. Here we have a dead pulp, but there is severe pain and great tenderness from inflammation of the peridental

membrane. It is obvious that nothing applied within the cavity of such a tooth will relieve the pain. Yet these are the cases that physicians often attempt to treat with oil of cloves, etc. If there is a cavity in the tooth, this should be cleaned out as thoroughly as possible in order to allow gas from the pulp chamber to escape easily. A loose or broken-down filling can sometimes be removed by the physician, and this will give great relief by allowing escape of gas. Other measures worthy of trial for relief of pain are painting the gum overlying the tooth with tincture of iodine, deep scarification of the gum with a sharp knife, and application of ice or hot water bags to the face, or a hot bread poultice to the gum. Hypodermic injection of morphine must be resorted to in some cases. Sometimes the symptoms occur in connection with a tooth that, at a glance, is seen to be beyond repair, in which case immediate extraction is the surest means of relief.

Fourth Stage. When a fluctuating swelling is felt, the pus should be allowed to escape as soon as possible by an incision in the gum. This is best performed by means of a sharp pointed knife. The blade is wrapped to within half an inch of its point with adhesive plaster to avoid cutting the lip, and the instrument plunged quickly into the point of greatest swelling. The abscess may be more thoroughly broken up, if the patient will permit, by inserting a grooved director in the opening and working it about. Packing or drainage material in the wound is unnecessary. An abscess occurring in connection with a tooth that cannot be saved should be treated by *immediate removal of the tooth*. Frequently pus will be discharged through the socket of the extracted tooth, making it unnecessary to lance the gum. After evacuation of an abscess, the wound may be washed out with warm salt solution or any mild antiseptic. Peroxide of hydrogen should be avoided, as it tends to cause spread of the infection through inability of the liberated gas to escape.

The instruments required for the above described measures

of relief include: a mouth mirror; a fine pointed flexible steel probe; a hatchet-shaped dental excavator; a pair of dental dressing forceps, with the ends of the blades bent at an angle to the shaft; a sharp pointed knife; a small rubber bulb syringe; and two pairs of universal tooth-extracting forceps. These instruments should be possessed by every hospital.

Another dental condition frequently seen in hospital patients is *salivary calculus* or *tartar*. This is a hard deposit lime salts about the necks of the teeth, and has nothing to do with dental caries, being rather a protection than a favoring cause in this respect. Its harm lies in the fact that it fosters the growth of bacteria, producing a septic mouth, and leads to irritation and inflammation of the gums. Deposits of tartar are frequently associated with pyorrhœa alveolaris, or suppurative inflammation of the dento-alveolar ligament, which finally results in loosening and dropping out of the teeth. In the treatment of these conditions the mere prescribing of a mouth wash is of little value. To be of any benefit, the irritating particles of tartar must be removed. This can be done thoroughly only by special instruments in the hands of a trained dentist. It is hoped that before long all of our large hospitals will follow the example of The Philadelphia General Hospital in having a graduate resident dentist. Recent graduates could be obtained to serve a year without salary upon the same terms as the resident physicians. A resident dentist, in addition to caring for the teeth of patients in the hospital, would find ample work to do in the mouths of people applying at the dispensary to have teeth extracted. There is especially a wide field for the conservation of children's teeth in the neighborhood of this hospital, as the free dental clinics are so far away. In the absence of a trained dentist, the physician must make the best of mouth disinfectants. As a general mouth disinfectant and germicide, there is nothing that is so generally useful as tincture of iodine. In applying this, the particular

portion of the gum to receive the application should be dried, and the iodine also should be allowed to dry after its application.

I will speak briefly of some of the *systemic conditions resulting from oral sepsis*, and some of the *oral manifestations of general diseases*.

At the last meeting, Dr. Deaver spoke of chronic gastritis as being a very rare disease, in comparison to gallstones, gastric ulcer, gastric cancer, and other lesions giving symptoms referred to the stomach. However this may be, one of the most frequent causes of the lesions giving the symptoms grouped under the name chronic dyspepsia, is the constant swallowing of pus from a septic mouth.

So-called articular rheumatism, both acute and chronic, is very frequently caused by absorption of bacteria from foci of infection in the mouth. It is also recognized that this same cause may produce severe grades of anæmia, sometimes of the pernicious type, resulting in death.

The inhalation of infective material from the mouth into the bronchi during anæsthesia is a well recognized cause of postoperative pneumonia.

It is thus evident that the routine examination of the mouth in hospital cases will frequently reveal sufficient pathology to explain the disease on account of which the patient is admitted, and give an indication as to the proper treatment to be instituted. This is true especially in gastrointestinal diseases, in cases of arthritis, acute and chronic, and in so-called primary anæmias.

Of the systemic diseases having oral manifestations, syphilis is perhaps the most important. Primary, secondary, and tertiary lesions may occur in the mouth, so that examination of this cavity often affords valuable diagnostic information. It is most important too, in the treatment of this disease, that the mouth and teeth should be kept in a clean condition, thereby greatly lessening the danger of mercurial stomatitis. This can practically always be prevented by appropriate

prophylactic treatment before putting the patient on a course of mercury. All deposits should be removed from the teeth, cavities filled, and useless teeth extracted. During the entire course of treatment by the mercury a mouth wash should be used, and the teeth kept thoroughly clean. If stomatitis occurs, the mercury should be stopped, cleansing of the teeth should be carried out as stated above, and the inflamed gums painted with tincture of iodine; 1 to 2000 potassium permanganate solution is useful as a deodorant mouth wash.

Dental disease may give rise to acute or chronic enlargement of the cervical lymph nodes, especially of the submaxillary region, and it is always well to examine the mouth in these cases. Treatment cannot be considered thorough if diseased teeth are left as possible sources of infection, and removal of these is often all the treatment required.

Infection through the teeth is one of the commonest causes of so-called Ludwig's angina, or streptococcic inflammation of the cellular tissue of the floor of the mouth and neck.

Abscesses from diseased teeth occasionally open on the skin of the face and neck, leaving sinuses that may persist for months or years. The opening appears as a small red papilla through which pus can be squeezed. A probe passed through the minute opening will usually come in contact with bone. These cases are sometimes very puzzling to the physician who may make a diagnosis of lupus or some other chronic skin disease, and attempt to cure with all sorts of external applications. Removal of the offending tooth will cause the sinus to heal almost immediately. It is unnecessary to curette or drain the sinus after the cause is removed. Occasionally only the root of the tooth is present in the jaw, and may be covered by the gum, so that there is a question whether the sinus comes from dead bone. Here the x-ray is of the greatest value in diagnosis.

Symptoms that cannot be distinguished from true neuralgia major, or tic douloureux, are sometimes caused by lesions of the teeth, notably impacted teeth. An impacted tooth,

usually a wisdom tooth, is one that fails to erupt normally, but lies more or less horizontally, the eruptive force pressing it against the tooth in front. "Pulp-stones," or concretions forming within the pulp of a tooth, may also cause neuralgia. These conditions may be all beautifully shown by the *x*-ray. Indeed in any pathological condition of the face or jaws, in which the cause is not directly apparent, a good *x*-ray plate is of the utmost value.

Pathology of the teeth and mouth has many other bearings on the general organism at large, but the few points mentioned in the short space available, will give some indication of the importance of a thorough knowledge of this part of the body by the physician.

SOME CAUSES OF COUGH OFTEN OVERLOOKED, PERTAINING TO THE EAR, NOSE, AND THROAT¹

BY CURTIS C. EVES, M.D.
AURIST AND LARYNGOLOGIST TO THE HOSPITAL

I HAVE selected this subject because cough is one of the most troublesome and frequent conditions from which patients seek relief, and because we so frequently see patients having an annoying cough with no apparent cause in the chest, pharynx, or larynx.

Cough is a reflex action caused primarily by some irritation of the sensory fibres of the pneumogastric nerve, or due to mechanical irritation. It has for its object the removal from the air passages of obstructive or irritating materials. It may be excited by the irritation of nerves when there is nothing to be expelled. It is to this latter I wish chiefly to call your attention. There are, undoubtedly, some cases where quiescent tuberculous lesions of the lungs are lighted up by the irritation of coughing, originating from another cause.

Beginning with the ear, we find a number of patients with impacted cerumen in the external auditory canal complaining of an irritating cough and tickling in the throat. Occasionally this cough is severe enough to cause hyperæmia and congestion of all the upper air passages. The cause of this cough is due to pressure on the chorda tympani nerve which passes across the upper part of the membrana tympani, upon its inner sur-

¹ Read at a meeting of the Episcopal Hospital Clinical Society, March 18, 1912.

face. It then finds its way downward and terminates in the tongue on the same side. Often in simply wiping out the canal with cotton, the patient will cough or clear the throat. Dried secretion or a foreign body in the ear may give rise to the same symptoms. When the canal is cleared, the cough and irritation ceases.

A more common cause of cough is to be found in the nose, in which the posterior part of the inferior turbinates becomes relaxed (turgescient) or hypertrophied, so that the surfaces of the turbinates come in contact with the floor of the posterior nares or the mucous membrane of the epipharynx. The cough is set up by the simple irritation of contact of the mucous membranes. I have recently seen two cases, in private practice, of this kind, in which the cough completely disappeared when the turbinates were properly treated. To show how a simple irritation of the posterior part of the nares or postnasal space will cause coughing, we have but to introduce a cotton-tipped applicator gently, through the nose, into the postnasal space. Foreign bodies, cysts, mucous polyps, and other tumors in the posterior part of the nostril or epipharynx will cause cough.

Deformities of the interior of the nose rarely give rise to a cough, except when sufficient to cause mouth breathing. Adenoids are frequently a source in children of a croupy, barking cough. Hypertrophy of the lingual tonsil (an increase in the amount of lymphoid tissue found at the root of the tongue, back of the circumvallate papillæ) and lingual varix (a dilated varicose condition of the veins at the base of the tongue) cause mechanical irritation of the epiglottis, which gives the patient a feeling of fullness or of a lump in the throat, which he constantly tries to raise by coughing. In the latter condition the veins sometimes rupture and the patient expectorates blood or bloody mucus. Knowing as they do that cough and hemorrhage are symptoms of tuberculosis of the lungs, these individuals are much alarmed.

An elongated uvula by coming in contact with the tip of

the epiglottis, lingual tonsil, or touching the postpharyngeal wall lower down than usual, will frequently cause an irritating cough, which is usually relieved for a time by swallowing solids or liquids. Foreign bodies and growths of different kinds in the pharynx and larynx will all give rise at times to a cough.

Simple hypertrophy of the faucial tonsils rarely causes a cough, unless they come in contact with each other or touch the postpharyngeal wall. A cough noticed only upon lying down is usually caused by the enlarged posterior tip of the inferior turbinate, an elongated uvula, or hypertrophied tonsils.

By far the most commonly overlooked cause of cough in the throat is retained secretions in the crypts of the faucial tonsils. A patient may have a crypt, hidden by the tonsillar pillar, full of decomposed secretion or food particles, or the crypts may be in full view, yet keeping up a most persistent and annoying cough without any apparent redness or inflammation in the throat. Upon removal of the contents of the crypts, the cough disappears and you have a most grateful patient. Calculus of the tonsils rarely causes a cough.

Some individuals have a much more sensitive mucous membrane than others, and it is in this class of patients that the above conditions are found most annoying. All of these conditions are frequently seen in patients without the symptom of cough. On the other hand, it is always well to eliminate them when other causes cannot be found.

ENUCLEATION OF THE TONSILS AND REMOVAL OF ADENOIDS AND OF THE LINGUAL TONSIL BY SIMPLE METHODS¹

BY CURTIS C. EVES, M.D.
AURIST AND LARYNGOLOGIST TO THE HOSPITAL

It is now almost universally recognized that the entire removal of the tonsil tissue is the best procedure where operative interference is advised, and the attention of the laryngologist is intent upon finding a safe and simple method which can be performed quickly, leaving a clean and uninjured throat.

It is undoubtedly better for each operator to select the best method at his command and then to perfect the technique to the best of his ability. After one method has been mastered, it is comparatively easy to make changes which seem best to suit the operator.

Nothing new or original is claimed in the following method. It is a simple combination of steps used by many different operators, and history tells us that the American Indian chiefs used to pluck troublesome tonsils from the throats of their tribes by means of the fingers long before they were molested by a civilized medical profession.

Long anæsthetization for operations upon the tonsils always more or less jeopardizes the life of the patient who is affected with hypertrophy of the lymphoid glands, and oftentimes it is necessary to remove the tonsils of patients suffering from

¹ Reprinted from *International Clinics*, vol. iv, Twenty-second Series.

heart, lung, or kidney complications to whom an anæsthetic is harmful. The average time required for this operation is about five minutes for the removal of both tonsils and adenoids. Where time is of paramount importance to the patient's welfare, it can be done in less than two minutes under nitrous oxide, ethyl chloride, or primary ether.

I have used the following technique in a series of over seven hundred cases of all kinds of tonsils without any serious accidents:

General anæsthesia (ether) is preferred. After the patient is thoroughly under the influence, the anæsthetic is stopped. Further anæsthetization is rarely ever necessary.

The patient is then placed on the operating table in the horizontal position, with a small sand pillow underneath the shoulders, causing the head to incline slightly backward. The mouth-gag is introduced in the ordinary manner, the tongue depressor is placed well back upon the side of the tongue, and traction is made upon it downward and to the opposite side of the mouth, thus putting the outer pillar on the stretch and bringing it prominently into view. A right-angle curved knife is now introduced below and carried upward underneath the anterior pillar, between it and the capsule of the tonsil, separating the fold of mucous membrane reflected from the pillar upon the capsule (*plica tonsilaris*) and any adhesions that have formed between the anterior pillar and the tonsil; the incision is terminated in the supratonsillar fossa (Fig. 100). A knife with the opposite curve is now introduced with back uppermost into the top of the incision and gently held above and pushed backward over the superior pole of the tonsil; it is then rotated inward, cutting between the upper part of the posterior pillar and the capsule (Fig. 101).

The ball of the index finger of the left hand is placed in contact with the capsule upon the superior pole of the tonsil underneath the junction of the two incisions, and by gentle pressure downward, together with a rotary motion outward and downward, the finger forces the tonsil in its capsule out

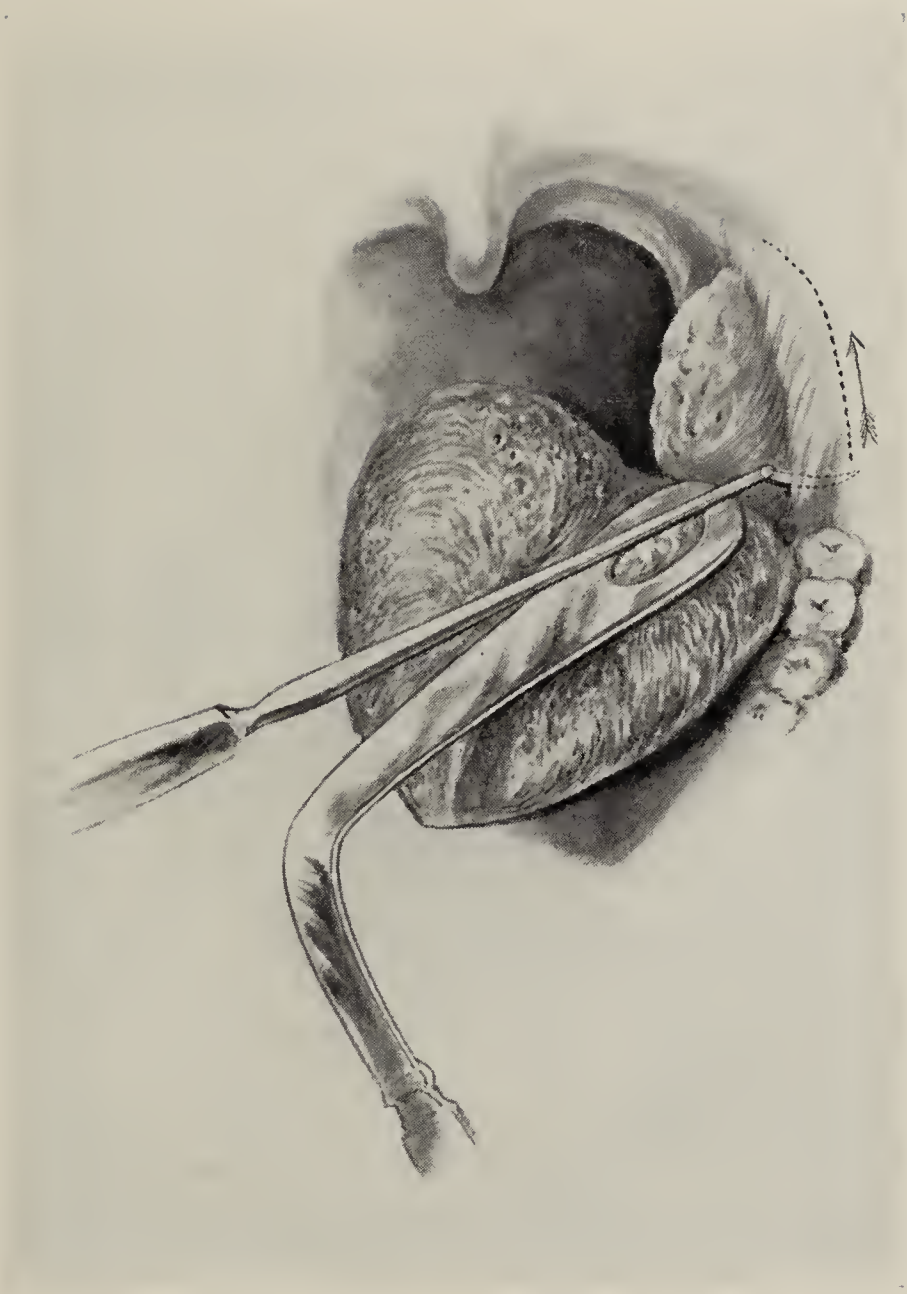


FIG. 100.—The right-angular knife introduced underneath the anterior pillar preparatory to making the incision upward. The incision is terminated by rotating the knife inward, bringing the end out in the supra-tonsillar fossa.



FIG. 101.—The left-angular knife, separating the upper end of the posterior pillar from the capsule of the tonsil. The knife is introduced with the back upward, then rotated inward, making an incision about the length of the knife-blade.

of its fossa. This motion of the finger is kept up until the fibrous tissue holding the capsule is entirely separated except at the base, where it is usually much thicker and more firmly attached (Fig. 102). The tonsil now lying loosely in the throat, except at the base, is caught over the upper part by a tonsil forceps, and is held on the stretch by an assistant, while the operator slides the loop of the snare over the forceps and fits it around the fibrous base with the left forefinger, at the same time gradually tightening the loop with the right hand (Figs. 103 and 104). When the loop is adjusted, the left hand assists the right in making the cut through the base, and the tonsil is removed.

The pillars of both tonsils are separated with the knives before the finger is used, then the removal of one tonsil is entirely completed. If both tonsils are peeled out with the finger before either base is snared off they are very apt, especially if large, to fall inward, forming a valve-like closure of the larynx which may severely interfere with the respiration of the patient.

The field is kept clear of blood by the assistant with gauze sponges. As soon as both tonsils are removed the patient is turned upon the side with the head low to allow the blood to drain from the mouth. Very little bleeding usually occurs. If bleeding continues it is most often due to faulty technique, where the pillars have been cut, or when a piece of tonsil at the base has been cut through by not properly fitting the snare around the tonsil.

The instruments used are seen in Fig. 105.

The tonsil may be entirely taken out by the finger without the use of the snare, but usually so much force is necessary to tear through the fibrous tissue at the base that the reaction is much greater than by snaring through it.

The adenoids are removed, while the patient is turned upon the side, in the following manner:

The left forefinger holds the uvula and soft palate forward, the tip of the finger is placed in contact with the septum and

is used as a constant guide for the forceps (Fig. 106). The forceps are placed into the nasopharynx with the blades closed, in the median line, so that the end comes flush with the posterior margin of the septum. The blades are then opened, pushed firmly backward over the growth, closed tightly and with a twisting motion made to partly cut and partly tear the adenoid from its attachments. If any pieces remain they are grasped with the forceps and twisted off. This is followed by curetting the surface with the end of the finger wrapped with several layers of thick-meshed gauze saturated with pure alcohol. By this method the adenoid vegetation is thoroughly removed, injuries to the posterior tip of the Eustachian tube and stripping downward of the mucous membrane of the posterior pharyngeal wall are avoided. The alcohol disinfects the wound and considerably reduces the bleeding.

To complete the tonsillar groups in the throat we have yet to mention the lingual tonsil. It being the least considered and most often overlooked, I shall treat its hypertrophy a little more fully than the other two groups, of which only one method of surgical treatment has been considered.

The lingual tonsil is an ill-defined mass of lymphoid tissue found at the base of the tongue, behind the circumvallate papillæ, in front of the epiglottis. Normally, we see several small, irregular islands of lymphoid tissue about the size of a split pea on either side of the median line. When hypertrophied the appearance presented is not unlike that of the pharyngeal tonsil, except that it is of a brighter red color and more nodular. Only two or three of the islands may become hypertrophied, projecting backward in wart-like growths. Frequently the growth is covered with varicose veins, or the varicosities may occur unaccompanied by hypertrophy, when the condition is known as lingual varix. The causes of hypertrophy of the lingual tonsil are not well-known. It is, however, a well-known fact that hypertrophy usually begins about the time of life that the pharyngeal tonsils and adenoids begin

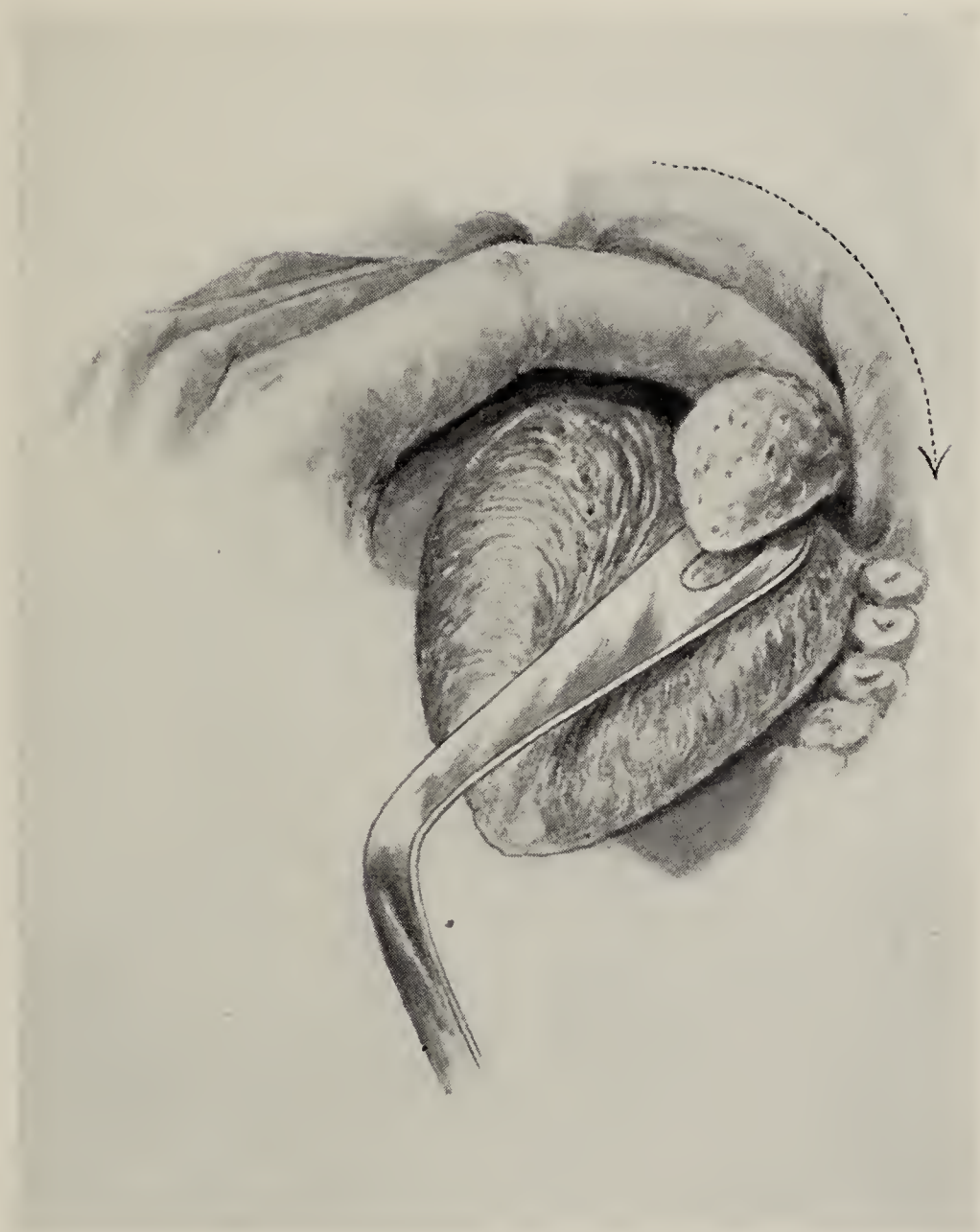


FIG. 102.—Finger peeling the tonsil in its capsule out of the fossa.

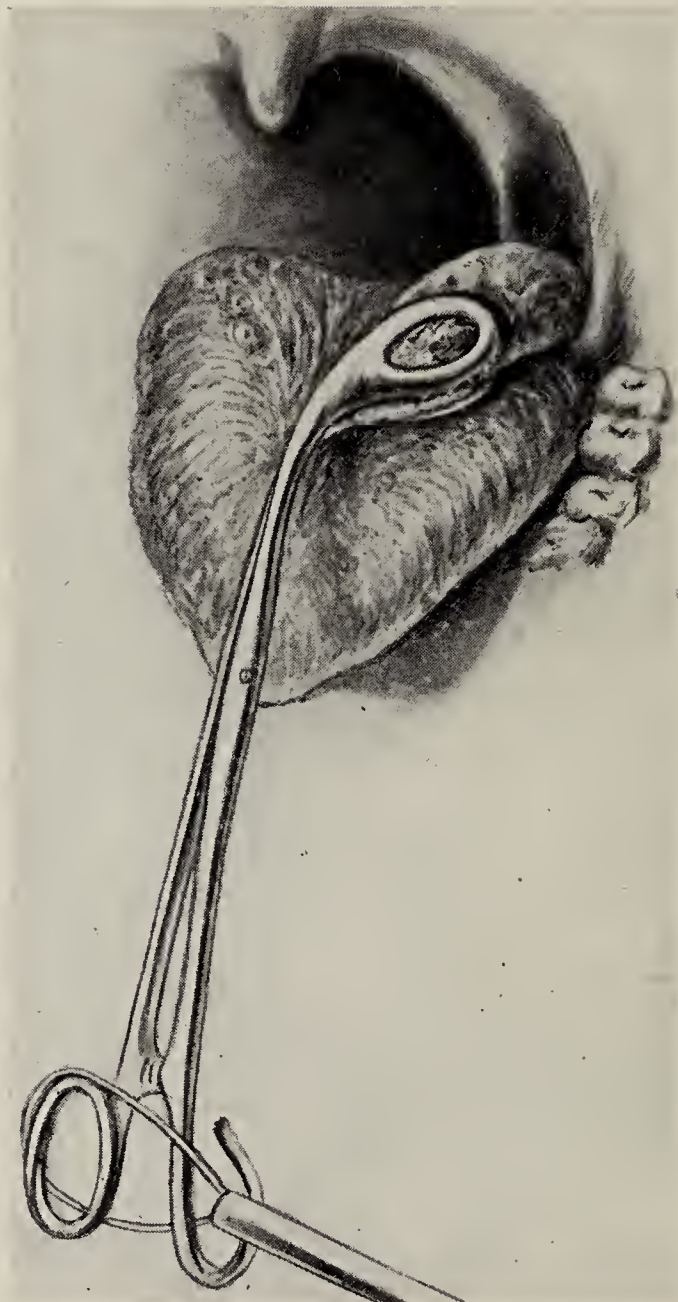


FIG. 103.—The forceps grasping the tonsil, and loop of the snare being passed over the handle.



FIG. 104.—Snare fitted around the base of the tonsil.

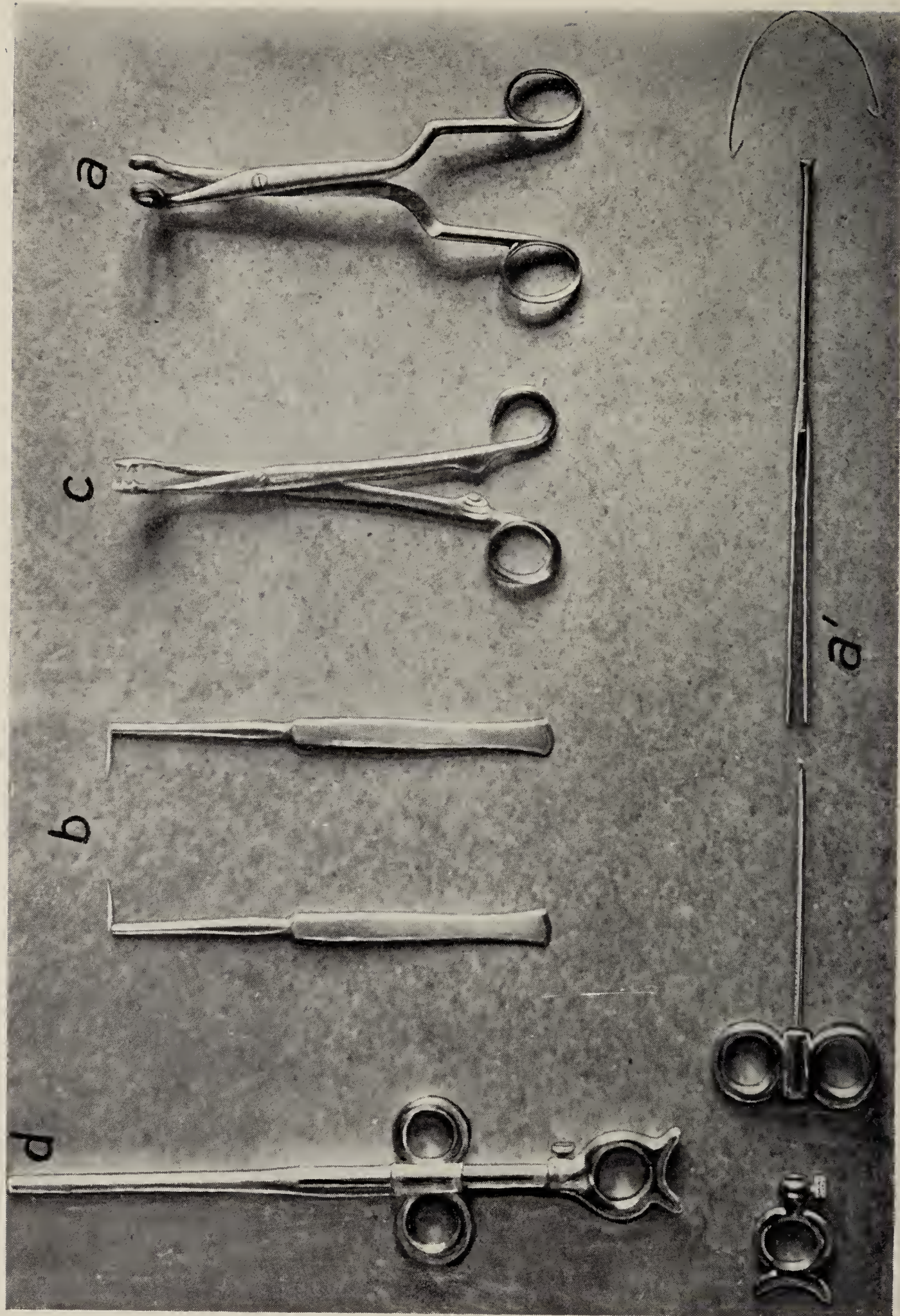


FIG. 105.—Special instruments used: *d*, Eves' tonsil snare; *a'*, Eves' tonsil snare in parts; *b*, Eves' modification of Leland's tonsil knives. The blades are ground shorter and rounded upon the points; *c*, Eves' tonsil forceps; *a*, French adenoid forceps.

to undergo atrophy, and that it does occur much more frequently in nervous, highly-strung females is an undisputed fact. Dudley, in his article published in the *Transactions of the American Laryngological, Rhinological, and Otological Society*, states that nine-tenths of the cases are seen in females after the twelfth year. He also says that trauma, rheumatism, gout, constipation and indigestion are predisposing causes. Lingual varix is most frequently seen in elderly people, and it then rarely gives symptoms. A hypertrophied lingual tonsil pushes backward against the epiglottis and may press so against it as to entirely envelop the tip, obscuring it from view.

The most annoying symptoms are caused by mechanical contact with the epiglottis. They are,—a sense of fulness or lump in the throat (often erroneously interpreted as globus hystericus), a tickling sensation and a dry, irritating cough, worse when lying down. Patients are apt to complain of some interference with swallowing. In singers, hoarseness and loss of voice are common, due to overdevelopment or fatigue of the muscles of the larynx, caused by clearing the throat and coughing. Among the less frequent symptoms are reflex pains and asthmatic attacks. When laryngeal varix accompanies hypertrophy the cough is apt to be followed by bloody expectoration, due to rupture of some of the veins. These patients are usually much alarmed, fearing they have pulmonary tuberculosis.

The diagnosis is made and treatment carried out by use of the laryngeal mirror. The patient holds the tongue forward with a napkin between the forefinger and thumb of the right hand. The mirror is placed gently against the soft palate so as to bring the reflected image of the growth clearly into view (Fig. 107). The following treatment has given me the best results:

Where the symptoms are of short duration applications of 2 per cent. cocaine solution, followed by 30 per cent. argyrol in equal parts of glycerine and water, are made every other

day. Should the growth not become reduced in size and the symptoms subside after a few treatments where the growth is of a soft nature, it is cauterized with 50 per cent. silver nitrate. Two parallel lines are made, one on either side, through the most prominent parts of the growth, 10 per cent. cocaine being used beforehand. Care should be taken not to touch the epiglottis or allow any of the silver solution to trickle into the larynx.

When the growth is large and of a denser nature, surgical removal is imperative and is carried out as shown in Fig. 107. A 10 per cent. solution of cocaine is applied two or three times, the Miles lingual tonsillotome is carefully fitted over the most prominent part on one side and made to cut through by pulling the knife-blade outward, at the same time pushing the forked blade inward. The other lobe is removed in the same manner. Sometimes it is necessary to make more than one cut on either side. The tissue is more fibrous than pharyngeal tissue and requires some force to make the instrument cut through. The instrument should be held firmly against the base of the tongue until the full cut is made. Very little bleeding occurs. The surfaces are then painted with 3 per cent. silver nitrate. Where lingual varix occurs only the cautery should be used, and repeated ever four or five days for several times.



FIG. 106.—Removal of adenoids with the French forceps, showing the position of the finger and the forceps grasping the adenoid mass.



FIG. 107.—Method of removing the lingual tonsil by the Miles lingual tonsillotome.

MALIGNANT DISEASE OF THE LUNG WITH SPECIAL REFERENCE TO SARCOMA¹

By A. A. STEVENS, M.D.
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SECONDARY malignant disease of the lung or pleura is not uncommon. It may arise by metastasis from distant organs or by direct extension from adjacent structures. Secondary cancer of the lung may be associated with an initial focus in one of the abdominal viscera, as the liver or pancreas; but in a larger percentage of cases it results from the extension of a primary mammary, œsophageal, or thyroid carcinoma. Encephaloid or medullary cancer is the form that most frequently reaches the lung by metastasis. Secondary sarcoma of the lung most commonly originates in a primary growth in the marrow cavity of one of the long bones. The small round-cell form is most often noted.

Two autonomous formations approaching closely to both carcinoma and sarcoma in their morphological characteristics, and appearing in many cases as secondary growths in the lungs, are the so-called hypernephroma and the malignant deciduoma. Of 22 cases of hypernephroma collected from the literature by Wooly² there was metastasis in the lungs in 13. Although the primary growth in these cases may corre-

¹ Read before the Section on General Medicine of the College of Physicians of Philadelphia, March 25, 1912. Cases I and III, from the Author's service at the Episcopal Hospital. Reprinted from the American Journal of the Medical Sciences, August, 1912, No. 2, cxliv, 193.

² American Journal of the Medical Sciences, cxxv, 1903.

spond morphologically to carcinoma or adenocarcinoma, it not rarely affords metastasis of a purely round or small spindle-cell sarcomatous type. Rolleston and Marks,¹ Wooley,² Adami,³ and others emphasize this point. Malignant deciduoma, which genealogically is more closely related to carcinoma than to sarcoma, involves the lungs secondarily in nearly 50 per cent. of the cases, and in some instances is first manifested by coughing, pleuritic pain, hæmoptysis, and other symptoms referable to the chest.

Primary carcinoma of the lung is rare. In the records of the Breslau Pathological Institute, out of 9246 necropsies, in which there were 1000 cases of malignant disease, Pässler⁴ found 16 cases of primary carcinoma and 4 cases of primary sarcoma of the lung. Seydel⁵ among 10,829 autopsies made at the Munich Pathologic Institute found records of 1342 tumors, and of these 184, or 13.7 per cent., were in the lungs or pleura. In 83.2 per cent. the tumors were metastatic and in 16.8 per cent. the tumors were primary. Of the primary growths 11 were sarcoma of the pleura and 20 were carcinoma of the lung.

With other observers primary carcinoma has been more rare and primary sarcoma more common than the statistics indicate. Reinhard⁶ found only 5 cases of primary carcinoma in 8716 necropsies. Rolleston and Trevor⁷ in analyzing the postmortem records of St. George's Hospital from January 1, 1890, to July 8, 1902, comprising 3983 cases, discovered only 8 cases of primary malignant disease of the lungs. Of these, 3 were primary sarcoma of the lung, 2 were primary sarcoma of the bronchi, and 2 were primary carcinoma of the bronchi. There was no case of primary carcinoma of the lung itself. The apparent inconsistencies in the statistics may be attrib-

¹ Amer. Jour. Med. Sci., cxvi, 1898.

² Loc. cit.

³ Principles of Pathology, i, 747.

⁴ Virchow's Arch., 1896, Band cxiv, S. 191.

⁵ Münch. med. Woch., 1910, lvii, No. 9.

⁶ Quoted by Villanene, Russkij Vrach, October 30, 1904.

⁷ British Med. Jour., February 14, 1903.

uted in part to differences in nomenclature and in part also, without doubt, to the fact that some of the older writers incorrectly classified their cases. Indeed, as Mallory's¹ studies have shown, it may be very difficult or even impossible to make definite statements in regard to the cellular structure of many tumors without operative removal and immediate tissue fixation.

Primary sarcoma of the lung may spring from the lymphatic glands surrounding the bronchi, especially those in the roots of the lung, from the lymphatic vessels themselves, or from the subpleural or intrapulmonary connective tissue. Tumors developing within the lung from the connective tissue are most frequently of the spindle-cell variety, whereas those originating in the lymphatic vessels are commonly of the nature of endothelioma. Endothelioma of the pleura often invades the lung, and unless the growth is studied histologically in its entirety it may readily be mistaken for lymphosarcoma or carcinoma. As a rule, primary sarcoma is limited to one lung. It forms a large infiltrating mass, of variable consistence and of a grayish color, often mottled with hemorrhagic areas. Metastases, except to the mediastinal glands and tissues, are not common. In the 8 cases of primary sarcoma of the lung itself studied by Rolleston and Trevor,² remote metastases were noted but once. In primary cancer, on the other hand, secondary growths are found with great frequency in the various abdominal organs, and not rarely in the muscles.³

The dominant symptoms of malignant disease of the lung may be those of a progressive consolidation of the lung, of stenosis of the larger bronchi or trachea, or of pleurisy with effusion. Cough and dyspnoea are rarely absent. The cough varies in severity and quality, according to the size and location of the growth. It is especially marked when the

¹ Jour. Med. Research, 1905, viii, 113.

² Loc. cit.

³ Ménétrier, Art. Neoplasie, Bouchard's Patholog. Gén., vol. ii; Handford, Trans. Path. Soc., London, 1888, xxxix, 48.

larger bronchi are involved or the mediastinum is invaded and the trachea compressed. In the latter event the cough is likely to be paroxysmal and of a ringing quality, as in aneurysm of the thoracic aorta. On the other hand, when the tumor is confined almost entirely to the vesicular structure the cough may be very slight. There is also much variation in the degree of dyspnoea. When the trachea is compressed, or a large pleural effusion present, it may be intense and for a considerable period the only obtrusive symptom. Expectoration occurs in a large proportion of cases, but it may be slight or even absent. Stokes laid much stress upon the occurrence of a gelatinous reddish (currant-jelly) or brownish-black (prune-juice) sputum, evidently the result of the intimate admixture of mucus and blood; but sputum of this character also occurs in other pulmonary conditions. Occasionally, disintegration of the tumor gives rise to copious foetid expectoration resembling that of gangrene. In a number of instances the diagnosis has been made certain by the microscopic examination of tissue fragments in the sputum.

Hæmoptysis, from congestion of the tissues surrounding the tumor, or erosion of fair-sized pulmonary vessels, is present in the majority of cases, and may be profuse and persistent. Pain is inconstant. It is often quite severe, however, when the pleura is involved or the thoracic nerves are compressed. Recurring attacks of pleuritic pain are not rarely the first symptom to attract attention. Enlargement of the superficial veins of the chest and localized œdema sometimes result from invasion of the anterior mediastinum and occlusion of large venous trunks. More rarely, hoarseness and dysphagia arise from pressure upon the recurrent laryngeal nerve and œsophagus respectively. Enlargement of the glands above the clavicle or in the arm-pit is an important symptom, but it is quite exceptional in sarcomatous cases. The temperature remains, as a rule, normal; but not infrequently in the later stages moderate fever of a remittant type supervenes and persists until the end. Cachexia sooner or later develops in

most cases, and is of importance in differentiating tumor from aneurysm.

In the absence of pleural effusion, percussion usually reveals an irregular area of dulness, gradually increasing in extent. If the bronchi in the affected region are pervious, auscultation elicits bronchial breathing and broncophony. In the majority of cases, however, the bronchi are occluded, and, in consequence, both respiratory sounds and voice sounds are feeble or suppressed. When the root of the lung is involved and a main bronchus partially obstructed the respiratory sounds over a limited area may be loud and stridulous. The chest wall over the tumor is sometimes distended, but it may be retracted if there are extensive pleuritic adhesions. In tumors attaining large dimensions the heart and abdominal organs are often considerably displaced. In a case of sarcoma of the thorax cited by Rolleston¹ the liver was so depressed that its lower border was on a level with the umbilicus.

In a large percentage of cases, symptoms of pleurisy with effusion dominate the clinical picture. If the fluid is not bloody at first, it usually becomes so after two or three tapings. Microscopic examination of the sediment sometimes affords valuable diagnostic aid. A large number of cells exhibiting numerous mitoses, especially asymmetrical division forms, is in favor of malignancy (Rieder, Warthin, Dock). Rarely, secondary nodules have developed along the course of the aspiration wound.

The following cases illustrate the important features of sarcoma of the lung:

CASE I.—W. P., a male, aged twenty-four years, with a good family history. His habits had been regular and he denied venereal infection. He did not recall having had any of the ordinary diseases of childhood, with the exception of diphtheria at the age of six. After that illness he enjoyed unbroken good health until about four months before admission to the hospital, January 12,

¹ Diseases of the Liver, p. 19.

when following exposure he was seized with sharp pain in the right side of the chest. His family physician treated him for pleurisy, and in four weeks his symptoms entirely disappeared. He remained in the "best of health" until six weeks before entering the hospital. At that time he developed what seemed to be a severe cold. The symptoms were those of sore throat, with hoarseness, hacking cough, and the expectoration of frothy mucus. This condition continued unchanged for about three weeks, when he noticed one morning on arising that his face was swollen, especially over the right cheek. The swelling subsided during the day, but returned each morning. Subsequently the neck and right side of the chest also became swollen. He had slight dyspnoea, but no pain, and his appetite and digestion were good. On admission the patient's condition was as follows: The face, neck, and right side of the chest were markedly oedematous. The skin was pale, but the appearance was not cachectic. The tongue was slightly coated, pale, and flabby. The voice was husky; beyond slight congestion of the vocal cords, however, there was nothing abnormal in the larynx. There was slight cough at intervals, with the occasional expectoration of frothy mucus. The pupils were equal and reacted well both to light and accommodation. The eye-grounds, except for slight congestion of the retinal vessels, were normal.

The right side of the chest was nearly 4 cm. larger than the left at the level of the nipples. Respiratory movements were absent on the right side and the intercostal spaces were obliterated. The apex beat was in the sixth interspace, near the anterior axillary line, and was forcible. Vocal fremitus was entirely absent over the right side, both anteriorly and posteriorly. On percussion there was flatness over the entire right side, except anteriorly above the clavicle. The cardiac dulness extended in the fifth interspace about 5 cm. to the left of the midclavicular line. On the left side there was hyperresonance. No respiratory sounds could be detected on the right side, except near the sternoclavicular articulation, where the breathing was bronchial. There were no friction sounds or rales on the right side, and vocal resonance was extremely feeble. On the left side the breath sounds were much exaggerated and scattered moist rales could be heard. A soft systolic murmur was audible at the apex of the heart, and was not transmitted. The heart sounds at the aortic area were normal. The pulmonary second sound was somewhat accentuated. The liver dulness merged above into the thoracic flatness and extended

below about 5 cm. below the costal border in the midclavicular line, otherwise nothing abnormal was found in the abdomen. Neither on admission nor at any other time could enlargement of the glands in the axilla or above the clavicles be detected. The respirations were 24 to 28, and not labored; the temperature ranged between 99° and 100° ; the pulses in the radial arteries were synchronous, but that on the right side was distinctly weaker than that on the left; the urine contained a trace of albumin, but no casts. Examination of the blood revealed: Hæmoglobin, 65 per cent.; red blood cells, 3,872,000; white blood cells, 13,400.

January 13. The right pleural cavity was tapped at two points: first, about 3 cm. below the angle of the scapula, and then 6 cm. below the angle of the scapula, near the posterior axillary line. Each puncture brought away about 50 c.c. of bloody fluid, which soon coagulated. Much resistance was offered to the entrance of the needle, and unless the direction of the cannula was frequently changed the flow of blood would cease. The clinical diagnosis was intrathoracic growth, probably sarcomatous.

January 15. The patient's condition was not materially changed.

January 16. The physical signs were unchanged. The patient stated that his appetite was good, that he had slept well, and was very comfortable. A blood examination revealed 15,600 leukocytes, of which 68.5 per cent. were polymorphonuclears, 29 per cent. were lymphocytes, 2 per cent. were large mononuclears, and 0.5 per cent. were eosinophiles.

January 17. The patient, against orders, got up and went to the toilet at the end of the ward. Returning he fell across the bed in a state of collapse, and died within a few minutes.

Autopsy. Body of a well-nourished man; skin and mucous membranes pale; rigor mortis (ten hours) well marked. Heart much displaced to left side. Left pleural sac was without adhesions and free from fluid. The left lung was somewhat congested and œdematous, but otherwise normal. In the right pleural sac there were dense adhesions, with loculi containing bloody serum, the latter amounting in all to about 120 c.c. The right lung was large and firm. It weighed 2500 grams. On section it was found to be infiltrated throughout, except for a small portion of the lowest lobe posteriorly, with a dense mass of purplish-gray color. A few areas of softening about the size of a pea were noted. No tuberculous foci were present. Neither the peribronchial nor the mediastinal glands were enlarged. The pericardium contained a few cubic centimetres of straw-colored fluid, and was without

adhesions. The heart was slightly dilated and relaxed. It weighed 320 grams. The valves were normal. The abdominal cavity contained about 300 c.c. of clear fluid. The liver was slightly adherent to the diaphragm. It weighed 1450 grams. It was congested and apparently somewhat fatty. The spleen was congested and firm. The other abdominal organs presented no gross changes. No secondary nodules were discovered. Microscopic examination of the growth revealed a typical spindle-cell sarcoma.

CASE II.—A. C., aged thirty-four years, a clerk by occupation, with a good family history. He always enjoyed good health until seven months before he came under observation, when he developed pain under his left scapula. This was shortly followed by a cough, with mucous expectoration and occasional attacks of hæmoptysis. The blood was bright red in color. He sought advice in a medical dispensary and was treated for tuberculosis, but did not improve. On the contrary, his cough grew worse, his expectoration became mucopurulent and profuse, the pain continued, he developed night sweats and often felt feverish, and in three or four months lost twenty pounds.

On admission the patient weighed 118 pounds, and presented a somewhat cachetic appearance. The chest was long and flat. The left side was immobile and slightly depressed. There was no glandular enlargement. The apex beat of the heart was invisible. Tactile fremitus was marked on the right side and absent on the left side, except anteriorly above the fourth rib and posteriorly above the scapular spine. On percussion there was flatness on the left side of the chest, extending from the fifth rib anteriorly to the fourth rib in the midaxillary line, and the spine of the scapula posteriorly. Over the right lung there was hyper-resonance. On auscultation scattered dry and moist rales were heard over the right side of the chest and over the upper lobe of the left lung. On the entire right side and over the left side below the clavicle there was harsh, vesicular breathing. Over the flat area on the left side the breath sounds were scarcely audible. Examination of the heart revealed no abnormalities. The organ was apparently not displaced. The abdomen was slightly distended, but otherwise showed no changes. The temperature ranged between 97.5° F. in the morning and between 100° and 101.5° F. in the evening. The pulse was 90 to 110; the respirations were usually 24 to 30; the urine was free from albumin, casts, and sugar. An examination of the blood showed: Hæmoglobin, 70 per cent.; red blood cells, 3,440,000; white blood cells, 12,400. The sputum on re-

peated examinations failed to show tubercle bacilli. The left pleural sac was punctured on three occasions, with negative results.

During the next four weeks there was no decided change in the general symptoms, although the patient became distinctly weaker and more emaciated. The expectoration was often blood-streaked, but only once in this period was there any marked hæmoptysis. Dyspnœa, which was slight at first, was now evident, especially on exertion. The fever and sweats continued as before, but there were no chills. Another examination of the blood, and the last one made, showed: Hæmoglobin, 65 per cent.; red blood cells, 3,200,000 white blood cells, 14,600.

In the fifth week, during a severe paroxysm of cough, he brought up in the sputum two particles of solid matter, one about the size of a small cherry-stone, the other somewhat smaller. They were of a grayish-pink color, irregularly rounded, and firm. Upon microscopic examination these particles were found to be made up of a close aggregation of large round or slightly oval cells. The cytoplasm was abundant and the nuclei were deeply staining. There was very little interstitial reticulum. A diagnosis of intrathoracic sarcoma was now made. Three days after the expectoration of the fragment the patient was attacked with severe hæmoptysis, which continued intermittently for about six hours. Following this he was much exhausted. Four days later he had another copious hemorrhage, followed by collapse, from which he never rallied. Death occurred about eight months after the appearance of the first symptom.

Autopsy. The body was much emaciated; the pleura on the left side presented firm adhesions below and posteriorly. On the right side there were slight adhesions posteriorly. Both pleural cavities contained a small quantity (50 c.c.) of bloody serum. The left leaf of the diaphragm was enormously thickened and tightly adherent to the left lung and spleen. The aorta, heart, and diaphragm were firmly united by adhesion. The bronchial, mediastinal, and abdominal glands were all much enlarged, but firm. The right lung was congested and œdematous. On section the left lung, except at the apex, was found to be studded throughout with numerous pinkish-white nodules, varying in size from that of a pea to that of an egg. Most of these were firm, but two or three of the larger ones were almost gruel-like in consistence. The right lung was congested and œdematous. It contained no nodules. The heart, apart from the adhesions surrounding it,

showed no abnormalities. The peritoneal cavity contained no excess of fluid, but presented local adhesions in the neighborhood of the diaphragm, spleen, and left kidney. The right adrenal body was about the size of a small egg, and completely replaced by a soft, reddish-white growth. The latter also extended a short distance into the superior pole of the left kidney. The right kidney, stomach, pancreas, liver, and intestines were apparently normal. No other nodules were discovered. The brain was not examined.

Microscopically the nodules in the lung presented for the most part the same picture as the fragments found in the sputum during life, although in places the resemblance to sarcoma of the alveolar type was close.

Microscopic examination of the adrenal growth showed it to be a so-called hypernephroma, some areas presenting a typically adenocarcinomatous aspect, and others exhibiting just as definitely the characters of alveolar sarcoma.

CASE III.—G. A., a Greek, aged thirty-six years, a laborer by occupation. Owing to the lack of an efficient interpretation only an imperfect history could be obtained. He had been sick for several weeks, complaining of pain in the right side of the chest, dyspnœa, and cough without expectoration. During the six days that he was under observation these symptoms were still present. He was a man of medium height, decidedly pale, but not markedly emaciated. Dyspnœa was very pronounced, and his lips and finger-tips were blue. The temperature throughout was normal or subnormal; the respirations were between 24 and 26; the pulse ranged between 110 and 120; the urine was somewhat scanty, but otherwise not abnormal. An examination of the blood showed: Hæmoglobin, 60 per cent., red blood cells, 3,260,000; white blood cells 12,000. The physical signs were those of a pleuritic effusion on the right side. This side of the chest was immobile and appeared to be slightly distended, although the interspaces were not obliterated. Fremitus was absent and the percussion note was flat at all points below the clavicle in front and the spine of the scapula posteriorly. The breath sounds were inaudible. Over the left side of the chest there were a few scattered coarse rales, but otherwise only the usual evidences of compensatory action. The apex beat was diffuse in the fifth interspace, and slightly to the left of the midclavicular line. There were no murmurs. The lower border of the liver could be felt below the costal margin, but apart from this the results of the abdominal examination were negative.

Two punctures of the right pleural sac were made on different occasions; the first yielded a few cubic centimeters of serum faintly tinged with blood, and the second, about 80 c.c. of fluid decidedly hemorrhagic. The clinical diagnosis was pleurisy with effusion, probably tuberculous. Two days after the last aspiration pulmonary œdema suddenly set in and terminated fatally.

Autopsy. At the autopsy only the thoracic findings were of interest. The right pleural cavity contained about half a liter of bloody serous fluid. The pleural surfaces were firmly adherent over the uppermost lobe, both anteriorly and posteriorly. Below there were no adhesions except over the diaphragm. The pleura throughout was enormously thickened and covered with numerous cartilaginous nodules. The right lung was compressed, hyperæmic, and œdematous. In the middle lobe there were two yellowish-white nodules, having a diameter of about 3 cm.; in the lowest lobe there were a number of nodules ranging in size from a millet seed to a hazel-nut. The left pleural cavity was empty, and the left lung was hyperæmic and œdematous. The heart was pale and flabby, but otherwise not abnormal. The enlargement of the intrathoracic lymph glands could be detected.

Microscopically, sections of the pleura showed numerous anastomosing tubules lined with one or two layers of cubical epithelial cells, and separated by a rather dense network of connective tissue. Interspersing the latter were many round cells, with abundant cytoplasm, and deeply staining nuclei, somewhat like those in round-cell sarcoma. Microscopic examination of the nodules found in the lung revealed a wide-meshed net of delicate connective tissue, the meshes of which were filled with large round cells, having an epithelioid appearance. The cells were packed tightly in the acini, and were in immediate contact with the reticulum as in alveolar sarcoma. Many vessels were seen in the trabeculæ. The anatomical diagnosis was endothelioma of the right pleura with metastasis in the right lung.

The diagnosis of pulmonary malignant disease is often a matter of great difficulty, only to be arrived at by a careful collation of all the facts and a close observation of the progress of the case. Pulmonary tuberculosis, pleural effusion, or aneurysm is usually suspected. Not rarely the symptoms throughout are so indefinite that the true nature of the condition may only be determined post-mortem. The discovery

of malignant disease elsewhere, of course, furnishes an important clue. This clue is likely to fail, however, when the growth is sarcoma and is primary in the lung. Even when the pulmonary affection is secondary the symptoms of the metastatic tumor may overshadow the entire clinical picture and the primary focus escape recognition, as in one of the cases cited above. Occasionally the examination of scraps of tissue found in the sputum furnishes conclusive evidence. In this way the diagnosis was established in Case II. Wolff,¹ Claisse,² Ehrlich,³ Betshart,⁴ Feldt,⁵ Demorest⁶, and Cornil⁷ also cite instances in which the microscopic examination of tissue fragments from the sputum afforded decisive information. When pleural effusion is present, the character of the fluid, its rapid return after thoracentesis, and the cytological findings are sometimes suggestive. However, too much importance should not be attached to the occurrence of a slightly bloody exudate, since this is not uncommon in tuberculosis and pleurisy complicating chronic nephritis and other wasting diseases. In cases marked by consolidation of the lung, suspicion may be aroused by certain anomalies in the clinical picture, as the unusual location or distribution of the dull areas and the constant presence of blood in the sputum without tubercle bacilli. The fact must not be forgotten, however, that tuberculosis and malignant disease of the lungs not rarely coexist. Tuberculosis was present in 13 of 31 cases of malignant disease of the lung reported by Wolff⁸ and in 3 of 10 cases reported by Schwalbe.⁹ Hildebrand,¹⁰

¹ Fortsch. d. Med., Nos. 18 and 19, 1895.

² Lancet, March 11, 1899, p. 712.

³ Quoted by Hampeln, Zeit. f. klin. Med., 1897, Band xxxii, H. 3 and 4

⁴ Virchow's Arch., Band cxliii, H. 1.

⁵ Deutsch. med. Woch., 1903, xxix, No. 28.

⁶ Med. Rec., January 16, 1904.

⁷ Comp. rend. de Soc. Anat., January 27, 1905.

⁸ Loc. cit.

⁹ Deutsch. med. Woch., 1896, xii.

¹⁰ Ziegler's Beiträge, 1888, Band ii.

Seigert,¹ Ribbert,² and Ménétrier³ also cite cases in which tuberculosis and malignant disease were associated in the same lung.

In any case much importance is to be attached to symptoms pointing to stenosis of the air passages, although in tumors developing within the lung these do not appear, as a rule, until the disease is far advanced, and even when such symptoms are well marked the existence of aneurysm must often be debated.

Pulmonary syphiloma may produce signs closely resembling tuberculosis on the one hand or malignant disease on the other. In the absence of any positive evidence of tuberculosis or malignant disease, a positive Wassermann reaction should be regarded as an indication for recourse to specific treatment. Echinococcic disease of the lung and dermoid cyst may yield most of the symptoms of malignant growth, although both are even more rare than the latter. Of 1816 cases of echinococcic disease occurring in the United States and collected by Summer⁴ the lung or pleura was involved in 147. Apart from the presence of cysts elsewhere, the expectoration of hydatid membrane, and the data afforded by thoracentesis, there are no characteristic signs. Presumptive evidence, however, might be forthcoming in the presence of pronounced eosinophilia and the fixation of complement in the hæmolytic test. Exploratory puncture is not without danger, owing to the grave toxæmia which may follow absorption of the hydatid fluid. Maydl⁵ reports 11 cases of intrathoracic echinococcus disease in which a fatal result followed thoracentesis.

In the case of intrathoracic dermoid cyst the coughing up of hair is the only pathognomonic symptom. According to

¹ Virchow's Arch., 1893, Band cxxxiv.

² Deutsch. med. Woch., 1896, xi.

³ Gaz. Heb. de Méd. et de Chirurg., 1899, No. 6.

⁴ New York Med. Jour., August 22, 1896.

⁵ Ueber Echinokokkus der Pleura, Wien, 1891.

Shaw and Williams¹ this was observed in 7 of the 35 authentic cases reported in the literature up to 1905.

Except in rare cases the treatment of tumors of the lung or pleura can only be palliative. In the 31 cases analyzed by Seydel there were only 7 in which an operation would have offered the slightest chance of recovery.

¹ Lancet, November 4, 1905.

THE UNCERTAINTY IN DIAGNOSIS OF UPPER ABDOMINAL DISEASES AND CONDITIONS¹

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IN these days, when the cry everywhere is early diagnosis; and as prognosis, especially in surgical conditions, depends in many cases upon that early diagnosis, it behooves us to try and conquer some of the obstacles that stand in our way.

There is probably no region of the body where we have so many obstacles to contend with in making an accurate and early diagnosis, as in the diseases of the upper abdomen. The main conditions, that is, the more common ones, which we must diagnosticate with certainty if we are to protect the patient both as to prognosis and result, are: (1) Cholecystitis; (2) duodenal ulcer; (3) gastric ulcer; (4) acute and chronic pancreatitis.

In the last few years much has been written on the so-called classical symptoms and the differential diagnosis between these conditions. The research and practical treatises on this subject have not been without their good results, but as we have gone further into this subject of upper abdominal disease, we have found ourselves at a loss for pathognomonic signs and symptoms, whereby we may differentiate with accuracy between them.

There are many reasons for the intermingling of the symptoms of upper abdominal conditions. In the first place, the

¹ Read before the Episcopal Hospital Clinical Society, September 16, 1912.

anatomical position of the pancreas, gall-bladder, pyloric end of the stomach, are all held in close approximation to each other. A silver dollar would easily cover the tip of the gall-bladder, the pyloric end of the stomach, and a portion of the head of the pancreas. There is also a distinct physiological connection between these organs. Besides these anatomical and physiological conditions, we also have many pathological conditions taking place in the upper abdomen, which all tend to embarrass us further in making an early and accurate diagnosis.

In 40 to 46 per cent. of both gastric and duodenal ulcers we have adhesions either to the gall-bladder, liver, or pancreas. With these adhesions to the neighboring organs and structures we are bound to have a variety of symptoms in different cases, which might easily lead to an uncertain diagnosis.

At least 75 per cent. of ulcers of the stomach are in the region of the pylorus, or in the lesser curvature in the neighborhood of the pylorus. As I have said before, 40 to 46 per cent. of both duodenal and gastric ulcers show adhesions, and in 75 per cent. of cases these adhesions are attached either to the gall-bladder or the pancreas; then again, in 20 per cent. of cases we see at least two or more ulcers in the stomach. In a very small percentage of cases we see an ulcer in both the stomach and duodenum at the same time.

When we stop to think of this wonderful complication of affairs taking place in the upper abdomen, is it any wonder that the surgeon feels that the diagnosis of these conditions is many times an uncertain one?

In the last few years many laboratory tests have been given us to try and clear up our hazy diagnosis. These laboratory findings, while in many cases of great value, have been on the whole misleading. The examination of the stomach contents is not without its pointings, and in many cases is of practical value in summing up our diagnostic data. But, on the other hand, it is many times misleading, and those who rely entirely upon such examinations often are misled

and many times leave their patients high and dry upon the rocks, like a shipwrecked vessel, so far as future surgical interference goes.

Graham, in his paper on the examination and practical value of the stomach contents in gastric ulcer and gastric carcinoma, in a series of 250 cases, reports the following:

In the gastric ulcer he found a total acidity below normal in 28 cases, normal in 106 cases, and above normal in 103 cases. The free hydrochloric acid he found below normal in 23 cases, normal in 102 cases, and above normal in 112 cases. He claims that three-quarters of the cases of gastric ulcer that come to the operating table do not show high acidity, as is commonly thought to be the case. He found free hydrochloric acid in 70 cases in carcinoma of the stomach, and found it absent in 80, in a series of 150 cases.

This same deviation from the straight and narrow teachings is true of the examination of the stools, and of the Cammidge reaction. Occult blood in gastric and duodenal ulcers is found in from 13 to 14 per cent. of cases only, in the reports of different authors. It is also seen in a great variety of other conditions, as pointed out by Finney.

The Cammidge reaction, while seemingly of great value in the diagnosis of pancreatic disease, in the hands of Robson and Cammidge, has not given such uniform success in the hands of American surgeons.

Many other tests have been given us to help solve the difficulties of upper abdominal diagnosis. Suffice it to mention a few of the more important ones:

Schmidt's, for observing the digestion of beef muscle fibres.

Müller's test, for demonstrating the absence or presence of a proteolytic enzyme in the fæces.

Sahli's desmoid test.

Salomon test, for the amount of lecithin in the fæces.

All these tests have helped to solve some of the problems, but they are still in their infancy and not without their shortcomings.

I do not mean to condemn laboratory methods, but too many have a tendency to delay surgical interference, when it is most advantageous, because our laboratory report does not coincide with our history.

We have many conditions resulting from pathology elsewhere in the body that mimic upper abdominal conditions to a marked degree. First and foremost is chronic appendicitis. In some cases we see chronic appendicitis mimic the symptoms of upper abdominal disease to such a strong degree, that while in many cases it can be diagnosticated with accuracy, it is almost impossible to conceive that an appendix could produce so much upper abdominal distress. It will not only give you, in many cases, the so-called cardinal symptoms of ulcer of both stomach and duodenum, but will also give you, in some cases, some of their laboratory findings. Chronic appendicitis will also give you symptoms so strangely suggestive of gall-bladder disease, that it would be impossible to exclude gall-bladder disease in summing up your diagnosis. The author has seen a case of chronic appendicitis accompanied not only by jaundice, but by clay-colored stools, and yet on opening the abdomen, has found an apparently normal gall-bladder.

If we do not diagnosticate our cases early, what further pathological conditions or risks do we expose our patient to?

Graham has shown us that at least 70 per cent. of cases of carcinoma of the stomach have their origin in an old ulcer scar. Following ulcer of the stomach and duodenum we also see dilatation and contraction of the stomach, with their resulting deformities, such as hour-glass stomach, pouch formation, and the like. Another danger is that of perforation of both gastric and duodenal ulcer.

Furthermore, I think it is now safe to say that without gall-bladder disease we would never encounter gallstones, and we would rarely see either acute or chronic pancreatitis if it were not for gallstones. We never encounter the so-called cardinal symptoms of gall-bladder disease until complications

set in, and it is these complications of gall-bladder diseases that give us our cardinal symptoms whereby we make our diagnosis. It may be also safely said that, in the majority of cases, both carcinoma of the gall-bladder and bile ducts and carcinoma of the head of the pancreas are directly or indirectly the result of either a diseased gall-bladder or gallstones.

What are we to do then to prevent the occurrence of these conditions, which make, in some cases, the application of surgical treatment only a palliative one? No one appreciates more than the practical surgeon how fruitless it is to diagnose with accuracy these conditions. There are few, if any, symptoms or pathognomonic signs that can be relied upon to make our diagnosis in all cases an accurate one.

Dr. John B. Murphy has this to say in the diagnosis of some of the intrinsic but common upper abdominal conditions: "When you come to analyze cases of gastro-intestinal lesions, and particularly lesions of the stomach and duodenum in connection with lesions of the gall-bladder, the three common lesions of the upper quadrant of the abdomen, you must take into estimate certain things. First, pain; second, and only very little less in significance, gaseous eructations; third emesis, and then hæmatemesis, or the passing of blood in the stools; fourth, the local sensitiveness on pressure over the ulcer zone, and fifth, distress or relief produced by the ingestion of food. These represent the fine elements that must be weighed in making a differential diagnosis as to whether it is gall-bladder lesion, a duodenal ulcer, whether it is gastric ulcer, whether it is a reflex from the appendix or from the pelvic zone, or from some other place, or from the central nervous system; that is, syphilis, which gives its gastric crisis. All of these, if they balance, if they come with each symptom in an almost uniform way, the case is then typical. On the other hand, if they vary, then you can make a large interrogation point on the abdomen before you open it, and cut through to the diagnosis."

Moynihan, in his last article on acute pancreatitis, tells us that we will never be able to reduce the mortality of acute pancreatitis until we have a pathognomonic symptom or physical signs whereby we may diagnosticate it early and with accuracy.

About the only symptom that we have in common in all these conditions is pain, varying in intensity from mere epigastric distress to agonizing pain. There are many of us who do not as yet seem to appreciate the significance of abdominal pain and prefer to wait for cardinal symptoms. Abdominal pain always means pathology. That pain does not have to be severe and agonizing, as in many cases and over a long period of time it only shows itself as a continuous *distress*. Pain should always be looked upon with suspicion regardless of the general appearance of the patient, or in the absence of the more classical symptoms. We must first of all impress upon our minds that gallstones, ulcer of the stomach and duodenum, and pancreatic disease, are not rare diseases. They are indeed very common, even more common than probably the most enthusiastic explorer of upper abdominal conditions has any idea of.

Among the many relics of antiquity is the idea that both acute and chronic gastritis are much more common conditions than ulcer of the duodenum and stomach, gall-bladder and pancreatic disease. Chronic gastritis, as a primary condition, is very rare indeed. The great majority of cases of chronic gastritis are secondary to primary disease elsewhere in the body, following both chronic heart and pulmonary conditions.

This idea has a great deal to do with bringing our cases late to the operating table. The symptoms of acute and chronic catarrh are usually so distinctive that little doubt can exist regarding the nature of the attack. These are not by any means common conditions, and when compared with gastric and duodenal ulcers and gall-bladder disease, may be said to be rare indeed.

Many of our best hospital reports still show among their list a great number of cases of acute and chronic gastritis, gastro-duodenitis, etc. In most hospital reports, especially in dispensary reports, these conditions far exceed such diagnoses as gallstones, gastric and duodenal ulcers, and chronic pancreatitis. I feel sure that if it were only possible to follow these cases for a certain length of time, that most of them would come to the operating table, and when the abdomen was opened, to our surprise, we would find a diseased gall-bladder or appendix, a duodenal or gastric ulcer, or a hard pancreas, the cause of the patient's long continued "dyspepsia."

I feel sure that if all our cases showing upper abdominal distress were to go to the operating table, we would in many cases be less prone to rely on chronic gastritis for our diagnosis.

Most of our later pathology volumes tell us that chronic gastritis is still an uncommon condition to find at the post-mortem table, especially so when we consider its frequency to the finding of the so-called "innocent gallstones."

I feel sure that most of these snap diagnoses of acute and chronic gastritis depend in a good many cases on a too healthy reliance upon our laboratory data, and a failure in most cases to go back in our patient's life and retrace the various stages of his dyspepsia or bilious attacks.

Another idea that tends to baffle our already hazy diagnosis is the idea that some gallstones may be called innocent. This is due to the fact, I believe, that gallstones many times have their symptoms ameliorated, or at least temporarily controlled, by appropriate medical treatment. This is indeed unfortunate for the patient, because the long continued but neglected disease at last produces myocarditis and other cardiac lesions; and if it were not for these serious visceral complications I feel sure that our mortality in gall-bladder surgery might be said to be *nil*.

Many other abdominal conditions may also be improved temporarily by proper dieting, rest, and appropriate medical

treatment, such as the administration of various stomachics. It is a lamentable fact that some physicians will prescribe such treatment in upper abdominal conditions, and will go ahead for years treating their patients symptomatically, instead of looking for the cause of such symptoms. Throughout this long term of treatment pathology is going on, even though the patient may be benefited from time to time, and it should not be forgotten that the reputation of the practitioner is especially involved by hesitation in a case where ignorance must in the end be so palpably apparent.

There never has been any medical treatment instituted that has cured either gallstones, gastric or duodenal ulcer, or pancreatic disease, and the more we see of pathology in the upper abdomen, the more we feel certain that there never will be. Statistics would seem to indicate that more than 35 per cent. of upper abdominal conditions, untreated surgically, are fatal.

These conditions have no more place in medicine than appendicitis occupies today, and when the profession educate the laity along this line of thought, just so soon will we be able to stop furnishing the pathological museums with pathological curiosities from the upper abdomen.

Statistics tell us that 18.5 per cent. of cases dying from hemorrhage due to gastric and duodenal ulcer die so suddenly that operation is of little avail.

We have pathology elsewhere in the body that gives us many uncertainties when we come to diagnose upper abdominal conditions, namely, reflex symptoms from nephritis, cardiac disease, pulmonary and abdominal tuberculosis. Neuroses of various kinds also give us symptoms in the upper abdomen, many times of a cardinal nature, and tend to confuse us with true abdominal disease.

What are we to do then to do away with the uncertainties in upper abdominal diagnoses? Are we to be egotistical enough to make a diagnosis in all these conditions in a certain percentage of cases? Are we to go on with these conditions of

upper abdominal distress in a haphazard way, giving our patients temporary relief with rest, proper diet, and medication? Are we to wait until our laboratory results coincide with our symptomatic data, and go on until it is too late for the proper surgical intervention?

The only way that we can come to an early diagnosis in these conditions I feel sure is by careful supervision of our patients with upper abdominal disease, especially those that have shown chronicity in their course. We should have gastric ulcer, duodenal ulcer, gall-bladder, and pancreatic disease always in our mind, and only think of gastritis, both acute and chronic, neurasthenia, and such like diagnoses, when we have eliminated the above conditions by a thorough physical and laboratory examination.

There are many symptoms to be thought of and dealt with separately in making our diagnosis in patients complaining of upper abdominal distress.

First of all, pain, varying in character from mere distress to dull pain, to pain of an agonizing character; whether regular or irregular in time; whether it is aggravated or relieved by the taking of food or entirely independent of it; whether it ceases abruptly or continues; the radiation of the pain should always be taken into consideration.

Next, vomiting, its presence or absence, its character and frequency; whether it precedes, follows or is coincident with the pain, and whether it is dependent or independent of food; the quantity and character of the vomitus; finally, the presence or absence of hæmatemesis.

Nausea: its degree, frequency, and whether associated with pain or taking of food.

Gaseous eructation: Whether continuous or paroxysmal, whether it is dependent upon the taking of food, and finally, its chronicity.

The health of the patient: Whether well nourished, anæmic, cachectic; whether hopeful and active, or depressed, languid, weakened, discouraged and pale.

Finally, our laboratory diagnosis as to the examination of the gastric contents, stool, blood, and urine.

Therefore, for the sake of the health of the patient, and that we may be able to give him a perfect result, we must strike a happy medium.

If an exploratory operation is ever justifiable, it is in upper abdominal conditions showing a chronic tendency, especially so when we can exclude with certainty the presence of gastric symptoms, due to the reflex phenomena of cardiac disease, nephritis, tuberculosis, and the various gastric neuroses.

It is true that with all these precautions, careful watching of our patients, and elimination, we will sometimes meet with setbacks, both as to diagnosis and result, but I feel sure that they will indeed be few.

On the other hand, if we wait for cardinal symptoms to make our diagnosis, we will in many cases open abdomens, as we do today, and close them with a feeling of unavailing regret.

When we learn to look upon all upper abdominal symptoms with suspicion, and to be always on the lookout for pathology in that region, we will then, and not until then, eradicate adhesions with deformity, gastric, pancreatic, and gall-bladder cancer, and finally place the surgery of the digestive system where it properly belongs.

SARCOMA OF THE VERMIFORM APPENDIX

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THE rarity of sarcoma of the appendix makes the condition one of particular interest from several points of view:

1. The origin of sarcoma of the appendix.
2. The type and the frequency, as compared with sarcoma of other parts of the intestinal tract.
3. The prognosis.
4. The clinical symptoms and diagnosis.

I. THE ORIGIN OF SARCOMA OF THE APPENDIX

It is very difficult to decide what group of cells in a particular area of tissue has been the starting point of a sarcomatous process.

In the few cases reported, the tumor has been comparatively small, and the diagnosis has been made only on the routine microscopic examination of the specimen. It has been difficult to state whether these tumors have accompanied an inflammatory process, or followed it; or whether they have resulted from an entirely independent process (sarcoma) that has later caused inflammatory change.

Sarcoma is classed as a connective tissue tumor; but unlike all other connective tissue tumors, the cells do not advance beyond their embryonic state, but multiply as such.

Difference in the predominating cell of the tumor has caused a subdivision of sarcomas into distinct types. It is the study of these cell types that gives the life history of the tumor. However, while some sarcomas are composed almost entirely of one type of cell, other tumors show an admixture of cell types. Beside this class of tumors, composed chiefly of simple connective tissue, the more highly specialized connective tissues may show similar sarcomatous growth, as in bone sarcomas.

2. THE TYPE AND FREQUENCY OF SARCOMA OF THE APPENDIX AS COMPARED WITH THE NEOPLASM IN OTHER PARTS OF THE INTESTINAL TRACT

The type of cell found in the reported cases varied, viz., small round cell, 8; spindle cell, 3; lympho-sarcoma, 1; fibro-sarcoma, 1; and our case, small round cell. These are all of the simple sarcoma types, and evidently had their origin in some part of the appendix. The striking prevalence of nine small round cell- and one lympho-sarcoma, out of a total of 14 cases, suggests strongly that the origin in these cases has been in the lymphatic structures so prominent in this organ.

The appendix is apparently far less frequently the seat of sarcoma than are other parts of the intestinal tract. The majority of intestinal sarcomas are at the ileocæcal valve. Here, also, as in the appendix, the tumor most often seen is of the round cell variety. This suggests, again, the lymphatic structures in these regions as the starting point of the sarcoma.

3. THE PROGNOSIS

The diagnosis of sarcoma of the appendix has been in every case reported that of the pathologist, based on microscopic findings. The tumors have been small and very early

in their development. In a few cases the tumor was situated near the cæcal end of the appendix, with involvement of the cæcum and draining lymphatics. In these, the prognosis has been grave, both as to recovery and recurrence. But in the majority of cases the distal end of the organ was involved, and widespread infiltration and metastasis had not occurred, and in these the prognosis has been remarkably good. In the case reported by Warren, he mentions that the patient was well and free from recurrence four years after operation. This is striking evidence of the slight malignancy of these growths as compared with those in other parts of the alimentary canal.

4. CLINICAL SYMPTOMS AND DIAGNOSIS

The duration of symptoms referable to the appendix, when mentioned in the cases reported, is stated as five years, thirteen years, one year, one month, three months, five months, one year, ten days, twelve months, five weeks, and in our case, five days. In every case they were those of acute or recurrent attacks of appendicitis.

One case reports a temperature of 102° , pulse 100; another, temperature normal, pulse 100; another, temperature normal, pulse 80 to 90; another, temperature and pulse "slightly elevated." No mention is made of temperature of the other cases. Our case was admitted with a temperature of 99.4° , pulse 120.

One case presents the blood picture as follows: Hæmoglobin, 70; erythrocytes, 4,000,000; leukocytes, 12,000. Another reports a leukocytosis of 14,000. Our case had a leukocytosis of 12,000.

An accurate diagnosis before operation, therefore, appears impossible. In every case the symptoms were those of some type of appendicitis, and inflammatory changes, either chronic or acute, have usually accompanied the growth.

In 840 appendices examined in the Pathological Laboratory of the Episcopal Hospital from January 1908, until the present time, one case of sarcoma has presented itself.

E. F., aged twenty-five years, woman, admitted to the Episcopal Hospital, September 17, 1909, in the service of Dr. H. C. Deaver. Diagnosis, appendicitis. Her chief complaint was abdominal pain and vomiting.

Personal History. Patient had never had an attack similar to the present one. She had always been well, but had always been troubled more or less by constipation.

Present Illness. Five days before admission the patient was seized with an attack of pain in the lower abdomen, but did not vomit. Pain disappeared, but returned two days later, when she came to the hospital.

Examination revealed pain and tenderness over McBurney's point; slight rigidity of the right rectus. No mass felt.

On admission, temperature 99.4° , pulse 120, respirations 88. Leukocytes 12,320. Urinalysis showed light cloud albumin, and few hyaline casts.

Operation. September 18, 1909. Incision over McBurney's point. Condition found, apparently subacute appendicitis. No adhesions. Appendectomy and invagination of stump. Incision closed.

Recovery uneventful. Patient discharged on twenty-first day after operation. At the present time the patient is perfectly well, her general health having been much improved since her operation.

Pathological Diagnosis. Gross appearance. Appendix about 7.5 cm. long. Vessels slightly congested. About normal thickness. External surface smooth, showing no evidence of adhesions. At junction of the proximal and middle third, the appendix shows a more or less circumscribed, rounded area, about 1.5 cm. in diameter. The enlargement was pale, and of rather firm consistency, and showed on its surface several dilated small vessels from the mesentery. Section of the mass showed it to consist of whitish material of firm consistency. The remaining portions of the appendix showed on section a normal appearance.

Histological Examination. Section through the enlarged mass showed the lumen of the appendix to be filled up with small round cells arranged in alveoli, and one small area circumscribed, and containing a few epithelial cells and mucus. Muscular coats for the most part are expanded so that the individual muscle

bands are separated by strings of lymphoid cells. This is particularly true of the outer longitudinal coat. Beyond the outer muscular coat the same lymphoid cells, arranged in alveoli, are to be found. The peritoneal coat seems to be external to this. Type of cell is the small lymphoid cell of moderately deeply staining nucleus, and small amount of surrounding protoplasm. The cells are densely packed in alveoli, separated from each other by small bands of fibrous tissue, which carry the bloodvessels. The latter have, for the most part, very thin walls. Elsewhere in the tumor blood may be seen coursing through rows of cells in the alveoli. Histological examination of other parts of the appendix shows a slight chronic inflammatory condition, with slight production of fibrous tissue in the submucosa. In one section there is a small collection of lymphoid cells that seems to be a metastasis of tumor cells from the original growth. There is no evidence of an acute inflammatory condition, nor does the chronic fibroid condition seem to be of recent origin.

In reviewing the literature, the following cases of sarcoma of the appendix have been reported:

CASE I.—Gilford. A woman, aged twenty-seven years, with symptoms of chronic appendicitis for thirteen years. Spindle-cell sarcoma of the appendix, which was adherent to the colon and ileum, neither of which was involved.

CASE II.—Warren. Boy with symptoms of chronic appendicitis, intermittent pain and fever for one month. Tumor at ileocæcal angle with involvement of the appendix. Round-cell sarcoma with glands extending to the root of mesentery. Simple appendectomy. Was well four years later.

CASE III.—Glazebrook. A colored man, aged fifty-five years, died of apoplexy. At the autopsy the appendix was found to be enlarged to the size of a pigeon's egg, and surrounded by dense adhesions. Fibro-sarcoma at the tip of the appendix, originating in the serosa, and invading all coats.

CASE IV.—Patterson. Man, aged thirty-nine years. Uneasiness and occasional pain in the right iliac fossa for three months. An acute attack with vomiting just before operation. A tender mass palpable. Diagnosis, chronic appendicitis. Operation, appendix found to be thickened and adherent. The wall of the cæcum thickened for about a quarter of an inch about the appendix. This thickened portion was removed with the appendix. Patient died

six hours later. Autopsy showed no signs of tumor elsewhere. Round-cell sarcoma involving all coats except serosa.

CASE V.—Carvardine. Woman, aged forty-five years, had symptoms of appendicitis for five months. Had severe pain in the right iliac fossa, attacks of diarrhœa, and severe colic; a deep-seated, tender swelling present. Operation showed appendix three and a half inches long, size of thumb, very hard, adherent, and peculiarly friable. Cæcum not involved, but the head, and a neighboring enlarged gland removed. Recurrence on the left side. Death nine months later. Round-cell sarcoma.

CASE VI.—Carvardine. Man, appendix removed for recurrent appendicitis. Central portion contains a concretion. At tip is situated a white colored tumor the size of a hazel-nut. Some neighboring glands enlarged. Round-cell sarcoma. Author calls it lymphosarcoma.

CASE VII.—Bernays (Reported by Kelly and Hurdon). Woman, aged twenty-nine years. Negative family history. Symptoms of chronic appendicitis for one year. A hard tumor palpable in the right iliac region. Temperature normal. Operation, appendix found free, distal portion normal, proximal portion large and firm, adjacent cæcal wall infiltrated for a short distance on one side. Complete excision of the cæcum. Recovery. Round-cell sarcoma involving all layers of the appendix. Two years after operation the patient showed a tumor in the abdomen which was considered to be a recurrence.

CASE VIII.—Stewart. Man, aged thirty-five years. In course of a year had three attacks of what was diagnosticated recurrent appendicitis. Each attack more severe. Pain, muscular rigidity, vomiting, pulse 100, temperature 102. Operation, appendix found in the ileocæcal fossa, buried in what were taken for adhesions. Appendix enucleated with the finger. Simple appendectomy. Spindle-cell sarcoma.

CASE IX.—Jones. Woman, aged twenty-six years. Father died of metastatic sarcoma of retroperitoneal lymph glands, resulting from primary growth in testicle. Four years previous to operation patient fell on upright post on a wharf, striking herself in the appendiceal region. Pain and tenderness persisted in the lower abdomen for two weeks. Few months later a similar attack came on spontaneously, and during the following four years she averaged an attack every six months. Began suddenly with pain in the lower right abdomen. Nausea, but no vomiting. Marked local tenderness with considerable prostration. Small, hard tumor

at McBurney's point, quite immovable, and very tender. Pulse 100, temperature normal. Hæmoglobin 70, erythrocytes 4,000,000; leukocytes 12,000; polynuclear neutrophiles 78 per cent. Diagnosis, recurrent appendicitis with probably a small circumscribed abscess. Operation, head of cæcum covered with friable adhesions, out of centre of which the appendix, also very friable, was enucleated. Simple appendectomy. Spindle-cell sarcoma originating in submucosa of appendix midway between base and tip. It grew outward, invading successively all the coats of the appendix until the serosa was involved. It then extended to the serous covering of the cæcum, probably because preëxisting adhesions had already bound the appendix in its whole length to the wall of the cæcum. Five days after primary operation, wound reopened and cæcum resected, the ileum being joined to the ascending colon by lateral anastomosis. Sections of the cæcal wall showed the tumor invading the serosa, with here and there groups of cells infiltrating the outer muscular layer. No tumor cells were found at a deeper layer in the wall of the cæcum. The mucosa of the appendix was everywhere involved, though considerable round-cell infiltration was present. Subsequent recovery of the patient was uninterrupted, leaving the hospital at the end of the third week.

CASE X.—Power. Girl, aged seventeen years. Five weeks previously patient suffered from fairly typical attack of acute appendicitis of mild character. Lost 15 pounds in five weeks. Temperature normal. Pulse 80 to 90. Sudden attack of general abdominal pain and vomiting on day before operation. No mass could be felt. Urine normal. Leukocytes 14,000. Polymorphonuclears 86 per cent. Operation, simple appendectomy. Diagnosis, small round-cell sarcoma. Complete healing in two weeks. Patient steadily lost in flesh and strength. General abdominal sarcomatosis appeared, and patient died ten weeks after operation.

CASE XI.—Davis. Man, aged fifty-one years, who had suffered for over twelve months with recurrent attacks of pain in the right iliac fossa. Operation apparently done at the close of a semiacute attack. The appendix was behind the cæcum, firmly adherent, and bound down by the results of previous inflammation. Cæcum apparently not affected. Simple appendectomy. Microscopic examination showed a small round-cell sarcoma. Report made five months after operation and patient was then perfectly well and had gained 14 pounds.

CASE XII.—De Jong. Male, adult. Simple appendectomy. Appendix contained a small fæcal concretion in its central portion,

while the tip presented a tumor the size of a hazelnut, which gave the appearance of a lymph gland. Microscopic examination showed a small round-cell sarcoma. Report evidently made shortly after operation.

CASE XIII.—Wright. Boy, aged seventeen years. Illness began with severe colicky pains in the abdomen, and vomiting. He had been constipated for about ten days, but the bowels had moved with aperients. A mass was felt extending across the abdomen at the level of the umbilicus. Also an indefinite mass in the right flank. Temperature and pulse rate slightly elevated. Operation showed an intussusception, commencing in the cæcum and extending to the transverse colon. This was easily reduced, and was seen to have begun as a partial invagination into the cæcum. Tip of the appendix about the size of the terminal phalanx of the thumb and white and glistening. Several enlarged glands lying along the inner side of the ascending colon. Cæcum, ascending colon, appendix, glands, and terminal portion of the ileum removed in one mass. Recovery delayed by subphrenic abscess and fæcal fistula, but the patient was in good health two years later. Examination of the appendix revealed a lymphosarcoma. Glands were also the seat of secondary growth.

CONCLUSIONS. 1. Compared with carcinoma of the intestinal tract, sarcoma is rare; and the appendix is apparently the least frequent seat of the neoplasm.

2. Diagnosis of sarcoma of the appendix cannot be made in the early stages from the symptoms elicited.

3. The prognosis is good if the tumor is small, and situated some distance from the proximal end of the organ, and when adhesions and involved glands are not present.

4. Sarcoma of the appendix is usually of the small round-cell type, which is generally of rapid growth, infiltrating, and tending to rapid metastasis.

5. When operating upon a supposed inflammatory condition of the appendix, and a more or less circumscribed, infiltrated and enlarged, non-adhesioned appendix without an inflammatory condition is encountered, the adjoining head of the cæcum and the draining lymphatic glands should be most carefully examined and their condition determined.

REFERENCES

- Gilford. *Lancet*, 1893, ii, 241.
Warren. *Boston Med. and Surg. Jour.*, February 24, 1898.
Glazebrook. *Virginia Medical Monthly*, 1895, p. 211.
Patterson. *The Practitioner*, 1903, p. 55.
Carvardine. *British Med. Jour.*, 1907, ii, 1771.
Kelly and Hurdon. *The Vermiform Appendix*, 1905.
Harte. *Annals of Surgery*, xlvii, 968.
Davis. *Jour. Amer. Med. Assoc.*, December 15, 1910.
Jones. *Surg., Gyn. and Obst.*, February, 1911.
Powers. *New York Med. Jour.*, January 7, 1911.
Wright. *British Med. Jour.*, July 22, 1911.

A CASE OF ACUTE SUPPURATIVE PERITONITIS OF UNKNOWN ORIGIN, SIMULATING MENINGITIS ¹

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M. E., a white girl, aged six years, was admitted February 1, 1912, to the Episcopal Hospital, service of Dr. George W. Norris. When an infant she is said to have had two attacks of pneumonia; otherwise she had been well and healthy.

Present Illness. Patient is supposed to have been sick two weeks. Has been in bed one week. The character of the onset of illness is not known, except that the child first complained of abdominal pain and was quite constipated for a few days. Was given castor oil and since that time has had diarrhoea. Vomited a few times when first taken sick. Has been delirious for one week. Cries out continually with pain. No history of chills. No history of nose bleed.

Examination. A rather poorly nourished girl, of six years. Is very restless and irritable and somewhat delirious, and seems to suffer some abdominal pain. She lies on her right side, with thighs flexed on abdomen. Skin is warm and moist and extremely hyperæsthetic; the child cries out whenever she is touched or moved. No eruptions. Slight muscular rigidity but no tremors. Her temperature is 101°, pulse 140, and respirations 24; pupils are dilated and equal and iritic reflexes are normal; there are herpes on the lips; tongue is moderately coated; no marked retraction of head and only slight rigidity of cervical muscles.

Chest. Expansion good and equal, lungs clear and resonant throughout. Heart sounds are regular and no murmurs are present.

¹ Read at a meeting of the Episcopal Hospital Clinical Society, May 20, 1912.

Abdomen. There is generalized rigidity but it seems voluntary. No distention. No masses palpable. The child cries whenever the abdomen is touched. No vaginal discharge noticed.

Rectal examination not made.

Extremities. Slight suspicion of Kernig's sign on the left; no Babinski reflex and no ankle clonus; patellar reflexes about normal. Child is incontinent, both as to urine and fæces.

Leukocytosis 36,960, with 96.5 per cent polynuclears. Lumbar puncture showed no undue pressure within the spinal canal, and only a few cubic centimeters of fluid withdrawn; this was bloody, due to puncture of vessel. Culture was not made.

Progress. The diagnosis of meningitis was made, and the child's bowels were partly opened by cathartics and enema.

February 2, 1912. Patient transferred to Medical Ward. Her temperature is 102.6° F., pulse 144, respirations 28. She is extremely restless, irritable and moderately delirious, and seems to suffer pain. She cries out continually at short intervals. General hyperæsthesia of skin is marked. There is slight rigidity of neck, and a suspicion of Kernig's sign. No paralysis and no tremor. There is no ankle clonus and patellar reflexes are about normal. She vomited two or three times during the day.

February 3, 1912. Today she seems to be more rational and seems to have less pain, but is decidedly weaker. General hypersensitiveness of skin is less marked. Abdomen has become more distended, with board-like rigidity, and child will point to her abdomen when asked to tell what pain she has. There is a dull percussion note in the flanks. Diagnosis of general peritonitis is plainly evident.

February 4, 1912. Child died at 1.30 A. M. from profound toxæmia.

Autopsy. Acute suppurative peritonitis and cloudy swelling of all organs; pleura and lungs normal, except for slight congestion; heart showed normal condition and relations; the whole peritoneal cavity filled with thick, creamy, yellowish pus, with exudate on all serous surfaces of organs; there were no adhesions other than such as might be expected in an acute inflammation. The organs were in their normal positions and no external evidence was discovered as to origin of pus. The appendix showed evidence only of external congestion. Tubes and ovaries negative. Intestines showed slight enlargement of Peyer's patches and solitary follicles, which were probably not excessive for age of child. No evidence of ulceration to be found. Spleen slightly congested. Meninges showed no evidence of meningitis. At no point in autopsy was

there found evidence of the origin of pus. Histologically, no evidence of ulceration found in the intestinal tract and no evidence of any specific process, either typhoid or tuberculous. Cultures taken from lungs, spleen, and from the abdominal pus showed mixed growth. Heart blood showed pure culture of *bacillus coli communis*.

This case presents two interesting features. First, the apparently primary infection of the peritoneum by the colon bacillus, and second, the early prominence of the cerebral symptoms, which for a time masked the abdominal lesion.

That there are acute forms of peritonitis in which no site of infection can be discovered is generally recognized, but that these can be termed "primary" raises doubt in the minds of many observers. Thus, Stone,¹ in an article on pneumococcus peritonitis, writes that it is not logical to recognize a primary form. "It simply means that the portal of entry instead of being obvious, as in a precedent pneumonia, is obscure." However, Cuff,² writing on the same subject, says that there are cases of peritonitis where careful search, both ante- and post-mortem, fails to discover the site of infection and these may be justly described as primary peritonitis. Holt,³ in 1903, recorded a case of a baby, aged six months, in which autopsy showed acute diffuse streptococcus peritonitis, without apparent site of infection. Martin,⁴ in 1906, records a similar case, aged nine years, of general streptococcus peritonitis, without apparent site of infection. Monks⁵ refers to a case of streptococcus peritonitis without any assignable cause. Dowd,⁶ in 1908, placed on record three cases of streptococcus peritonitis of unknown site of infection.

The incidence of general peritonitis of unknown site of infection is greater in children (two to fourteen years) than in adults, and greater in female than in male children. However, Cuff records five cases of primary peritonitis, all of which were adult patients. Carmichael⁷ states that of all cases of general peritonitis treated in children's hospitals from 5 to 10 per cent. are due to pneumococcus infection.

MODE OF INFECTION. In considering the portal of entry of the infecting organism in the so-called "cryptogenic" forms of peritonitis, it is advisable to classify the possibilities under three groups, *i. e.*: (1) by way of vagina and Fallopian tubes; (2) by direct penetration of the walls of the gastrointestinal tract; and (3) by way of the lymph and blood streams.

VAGINAL ROUTE. Because of the greater frequency with which general peritonitis attacks the female sex, many have searched the vaginal route with the hope of finding the site of the infection. Merten⁸ reports five cases of peritonitis in girls, aged two to eleven years, preceded by suppurative process in the genitals. On the genitals of three of the children traces of scratches were found; for this the oxyuris was probably responsible. The scratches became inflamed and ascending peritonitis followed in time. That the colon bacillus can escape into the peritoneal cavity beyond the oviduct without making any particular demonstration *en route* is maintained by Morris.⁹ Dr. Joseph A. Blake¹⁰ recalls several cases of peritonitis in children, where infection was traced to the Fallopian tubes, and those in whom the tube was removed got well, while those in whom it was allowed to remain died. However, this will not explain the mode of infection in male children.

INTESTINAL ROUTE. Again, because of the early distinctive gastro-intestinal symptoms, *i. e.*, vomiting and diarrhœa, others believe that the infection is primarily intestinal. Flexner found diplococci within the lumen of the intestine, in its walls nearly to the muscularis mucosæ, and within the peritoneum, and also in spaces where he believes the lymphatics furnish the avenue of transit into the peritoneal cavity. Jensen produced a fibrino-purulent peritonitis in rabbits by feeding them virulent cultures of pneumococci, and excepting an enteritis and necrosis of Peyer's patches, autopsy showed no visible perforation. Bond shows that infection travels through the intestinal wall when there is combination of the

distended gut, with retarded blood supply, of faecal culture media in the intestine, and virulent organisms. Dowd records a case of general peritonitis in a boy, aged ten years, where at operation no site of the infection could be discovered. The appendix, which showed no evidence of perforation, was removed and placed in alcohol. On careful examination the following day he was able to force a little bubble through its wall near the tip. This indicated to him that there was a thin place which had furnished exit to the infection.

VASCULAR ROUTE. Other observers consider blood infection as the most likely theory, because the peritonitis is diffuse from the beginning, without enlargement of mesenteric lymph nodes, and is associated with profound intoxication; and because, in the pneumococcus variety, the infection is usually widespread, affecting lung, pleura, and pericardium. Championing this theory, Barling¹¹ writes that in the experimental inoculation of animals with pneumococcus, the lesion produced depends on the natural resistance of the animal. The more immune species develop acute local reaction at site of inoculation; the more highly susceptible, a general septicæmia. Hence, the frequency with which the multiple lesions are found in children and their heavy mortality; in adults, though the diplococci are widely distributed, as determined by isolation from the blood, no infection takes place elsewhere. Bonnet records a case of a child who died of purulent peritonitis, and expresses his belief that the source of infection was a facial erysipelas in the mother, and Oppenheimer describes streptococcus peritonitis in a child as an accompaniment of a widely spread and most virulent erysipelas. Cuff reports two cases of primary pneumococcus peritonitis: in one pneumonia developed twelve days after onset of abdominal pain; in the other meningitis developed eleven days after laparotomy for peritonitis.

SYMPTOMS. If we may judge from the reported cases, primary peritonitis seems to have a symptom-complex which should in most instances lead us to an early diagnosis of the

disease. Usually without history of previous ill health, the patients are suddenly seized with severe generalized abdominal pain, followed by fever, vomiting, and diarrhoea. The diarrhoea may persist or, after lasting a day or two, may be followed by constipation. From the onset tenderness is present, but rigidity and distention are noticeably absent or moderate in degree, until later in the course of the infection, when the patient quickly becomes prostrated from the profound intoxication. Cuff states that extreme restlessness has been noticed often, but in his cases apathy was particularly marked. Wasting is early and marked. After the first day or two, except in the most acute cases where the resistance seems to be paralyzed, leukocytosis is high—30,000 to 40,000. Herpes and petechial rashes have been noticed.

DIAGNOSIS. The diagnosis, again, if we may judge from the reports of published cases, is not always easy. To quote Dowd, the patients "are much more likely to have associated cerebral symptoms, so that very competent observers are sometimes at a loss to know whether a given case is to be considered as primarily cerebral or abdominal." In 1906, Armand and Bowen¹² collected from all sources 91 cases, and Bowen¹³ writes that four of these were under direct observation, and in none was the necessity for surgical intervention recognized. The list of diseases for which primary peritonitis has been mistaken includes tuberculous peritonitis, gastro-enteritis, typhoid fever, pneumonia, appendicitis and meningitis.

The *prognosis* is grave. The treatment consists in early laparotomy and drainage, which offer the best chance for recovery.

In conclusion, I wish to thank Dr. C. Y. White for his kind assistance throughout the preparation of the paper.

BIBLIOGRAPHY

1. Harvey B. Stone. Johns Hopkins Hospital Bulletin, July, 1911.
2. Archibald Cuff. British Medical Journal, April 18, 1908.
3. Holt, quoted by C. N. Dowd. Annals of Surgery, December, 1908.
4. Martin. Annals of Surgery, December, 1906, p. 919.
5. Monks. Annals of Surgery, June, 1908, p. 904.
6. Dowd. Ibid.
7. Carmichael. British Medical Journal, September 18, 1909.
8. Merten. Münch. med. Woch., January 30, 1912, lix, No. 5.
9. Robert T. Morris. Jour. Amer. Med. Assoc., March 2, 1912, p. 601.
10. Blake. Transactions of the New York Surgical Society, October 14, 1908, and Annals of Surgery, 1908, xlviii, p. 935.
11. S. Barling. Practitioner, London, April, 1912, lxxxviii, No. 4, pp. 489 to 624.
12. Armand and Bowen. Lancet, London, 1906.
13. Bowen. British Medical Journal, September 26, 1908.
14. Flexner. Johns Hopkins Hospital Bulletin, 1895.
15. Jensen. Arch. f. klin. Chir., 1903, lxix.
16. Bond. British Medical Journal, 1906.

SUPRAPUBIC CYSTOTOMY FOR THE REMOVAL OF A HATPIN FROM THE BLADDER¹

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THE patient, a Polish man, aged twenty-four years, presented himself on September 12, 1912, in the Surgical Dispensary, in Dr. Ashhurst's service. He bore a note from his physician stating that a few days previously someone had pushed a hatpin up his urethra while the patient was asleep and that the pin was still in the urethra. Since that time his urine had been bloody, but he had suffered little discomfort.

The patient was referred to the Receiving Ward, where a steel sound was introduced into the urethra; this met with a metallic obstruction about the penoscrotal juncture. Ten ounces of blood-stained urine were passed, but apart from some pain during the act, the patient made no complaint. After admission to the hospital, in Dr. Frazier's service, further inquiry through an interpreter revealed the fact that because of some difficulty in urination several days previously the patient had himself inserted a hat pin (head first) into his urethra, in an effort to overcome the fancied obstruction; but the point of the pin had broken off in his fingers, and the remainder (about five inches in length) had escaped into the deeper urethra. The patient denied all venereal diseases, and there was no evidence of a stricture.

A skiagraph (Fig. 108), made the next day, showed the hatpin, with its head well within the bladder, and its broken point caught in the subpubic urethra. As the pin could not be felt in the patient's perineum it was decided that its removal could be accomplished most easily and safely by means of suprapubic cystotomy. This was done on September 13, by Dr. Kleinhans, with the assistance of Dr. Ashhurst.

¹ Read before the Episcopal Hospital Clinical Society, September 16 1912.

Operation. The patient being etherized, the bladder was distended by injecting about six ounces of saline solution, through a soft catheter, which entered without difficulty. A median suprapubic incision, two inches long, was made, the sheath of the left rectus muscle being opened and its fibres split longitudinally. The peritoneal fold was displaced upward, and the bladder opened just enough to introduce a finger; this encountered the head of the hatpin, and being hooked around it easily drew it up into the wound whence it was removed. The bladder was closed without drainage by two or three sutures of chromic catgut, and the anterior sheath of the rectus was sutured in the same way. A small wick of iodoform gauze was placed in the space of Retzius, and the skin margins approximated with silkworm gut.

Though the patient's temperature rose to 103° F., the following day, it rapidly reached normal. The urine was quite bloody for twenty-four hours, and slightly blood-tinged for five days. The gauze wick was removed from the suprapubic incision on the third day after operation, and no leakage from the bladder could be detected. The sinus was entirely closed at the end of a week, and two weeks after operation the patient was discharged in good health.



FIG. 108.—Skiagraph showing hatpin in the bladder.

PUERPERAL ECLAMPSIA ¹

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IN view of the frequency of the condition (estimated by Fry at 6480 annually in this country alone) and the high mortality attending it, eclampsia may well command our most earnest endeavors.

I will briefly review some of the problems concerned in its management. Although much research work has been done, the primary cause of the toxæmia has not yet been determined.

From a practical standpoint, three questions of great importance present themselves:

1. How shall we recognize the approach of a toxic danger line?
2. Shall the uterus be emptied in antepartum or intrapartum eclampsia?
3. If so, how?

In answer to the first question, we have all been taught that the kidneys are the organs to be watched, and that the quality and the quantity of the urinary output will give the earliest signs of a gestational toxæmia in the latter half of pregnancy. And yet, we know that a notable percentage of patients, perhaps 10 to 20 per cent., will not show the evidence of greatly diminished urinary secretion and albuminuria a sufficient time before the eclamptic seizure to be of practical importance. Those who have had considerable experience in dealing with these cases have had many jars over the

¹ Read before the Episcopal Hospital Clinical Society, May 20, 1912.

disparity between the urinary findings and the convulsions. It is no uncommon thing to have a pregnant woman show albumin in the urine, and to have marked swelling of the hands and feet without marked toxic symptoms, while on the other hand a convulsion is at times almost the first symptom in a previously healthy woman. Confronted with such facts it will be seen how unreliable is the weekly routine examination of the urine in perhaps 20 per cent. of the cases.

It, however, is not to be neglected, for in a great majority of cases it does show the kidney condition, and when it is supplemented by œdema, headache, epigastric pain, impaired vision, and diminished urinary secretion, we recognize that the convulsive stage is imminent.

Latterly, we have added to our prognostic resources the blood pressure test. It has been shown that an increased toxæmia is marked by an advancing blood pressure, so that we have in the blood pressure apparatus a quick, easy and fairly reliable means of recognizing the danger line for convulsive seizures. A blood pressure of 160 to 180 mm. of mercury is an indication for the most active treatment. I have seen convulsions occur when the blood pressure registered as low as 153, and am verbally informed by a colleague of a case in his practice where convulsions occurred at a pressure of 130. I think that without doubt this is very exceptional. On the other hand, I have had a patient with a pressure of 240 without convulsions.

In answer to the second question, namely, shall the uterus be emptied in antepartum or intrapartum eclampsia, pretty much all teaching is in the affirmative. It is obvious that if the toxæmia is due to the pregnant state, the sooner the pregnancy is terminated the better it will be for the patient, it matters not what theory may be entertained as to the primary cause. To the objection that convulsions do not occur in a small percentage of cases until after delivery, the answer is, that the poisons have accumulated to such a degree that delivery has not afforded instant relief.

On behalf of the expectant plan of treatment, it is argued that these patients are all bad subjects for any operative procedure on account of their profound toxæmia. They succumb readily to shock, and indeed it is impossible to tell how serious may be the lesions in the brain, liver, or kidneys after the occurrence of the first convulsion—a condition which so frequently presents itself at this hospital.

The rights of the unborn child, in this condition, are to be considered. The trend of modern obstetrics is in the direction of conserving the life of the child. It is obvious that the earlier the eclampsia occurs in the period of gestation, the less chance will the child have to live, whatever the treatment adopted. Moreover, these children participate in the toxic condition of the mother. The placenta and even the child's organs may show lesions, and there is a large mortality among the babies born alive; so that when the acute toxæmia develops as much as three to six weeks before term, their chances at best are so poor that they may be practically disregarded in considering the treatment. From two weeks to term the child's rights are to be considered, although the mother's interests are paramount. Happily the mother's interests are usually the child's interests.

How shall we empty the uterus? Since my entry into the profession it has been the fashion, among the progressives at least, to adopt some quick method of emptying the uterus (*accouchement forcé*) after a convulsion has occurred, or when it is threatened. The most popular method of accomplishing this, and one which I have employed for many years, is first to dilate the os by the Harris method, and then apply the forceps or do a version. The Harris method consists in the introduction into the cervical canal of first the thumb and one finger, then the second, third, and fourth finger, in turn, using them in the manner of snapping the thumb and finger, thus dilating the internal os first, and drawing down rather than pushing up on the cervix in the manner of introducing a cone or wedge. Of course, the canal must be opened

wide enough to admit the thumb and one finger, the whole hand being introduced into the vagina, before the method can be applied, and this may necessitate instrumental dilatation up to this point to begin with. Unless the patient is comatose, an anæsthetic is necessary. Where the cervix has been effaced and only the os is to be dilated, this can usually be accomplished in half an hour. When the cervix is unobliterated and rigid, such as is apt to be the case in an old primipara, the process is too tiresome and too tedious to be recommended. The fingers become excessively fatigued and benumbed, requiring change of hands; and each change adds to the risk of infection. Even in a carefully managed manual dilatation of the cervix one is liable to lacerate the tissues; moreover, there is a great temptation to apply the forceps before complete dilatation is secured, and so do injury to the soft parts. I have once seen the cervix torn completely away from the vaginal wall posteriorly by efforts at extraction with the forceps before the canal was sufficiently dilated.

As I have said above, patients presenting a rigid cervix and undilated canal, such as old primiparæ are often found to have, do not well lend themselves to this form of treatment. For such a patient, a vaginal hysterotomy, or so-called vaginal Cæsarean section—an operation first described by Dührssen in 1896—meets the indication for quickly emptying the uterus. It consists in making an incision in the anterior vaginal wall, separating the bladder from the vagina and from the cervix, pushing it up well under the pubis and dividing the anterior wall of the cervix and lower uterine segment in the median line, and if necessary the posterior wall in a similar manner, until the opening is large enough to permit the passage of the baby. It is spoken of by some authors as a not particularly difficult operation, but my experience with it in three cases leads me to the conclusion that it should not be undertaken by any one who is not well qualified to do vaginal surgery—nor without ample assistance.

The difficulties encountered are not so much the hemor-

rhage, which may be considerable from the spongy and cyanotic character of the tissues involved, as the friable cervix and the impossibility of drawing it down within the range of vision owing to the impaction of the head in it.

After the incisions are made the baby can be quickly delivered by either forceps or version. After delivery the clean fresh incisions are sutured and the end results in about all instances should be as good or better than a forced dilatation with perhaps laceration, requiring even a much greater time.

The indication for the operation is *eclampsia*, when a quick delivery is sought; where the cervix is rigid and the os undilated in a patient who has previously borne children, and whose vaginal canal is therefore dilated, especially where the convulsions occur before term. Its contraindications are a nulliparous vagina and a high-placed cervix.

Dr. Peterson of Ann Arbor states in a paper on this subject that "Dührssen has collected 112 cases of vaginal Cæsarean section for eclampsia with a maternal mortality of 15 per cent. Veit has a series of 33 cases of eclampsia where vaginal Cæsarean section was employed with only one death, and that from pneumonia, from which the patient suffered before operation."

The classical Cæsarean section presents, I believe, less technical difficulties in its performance than does the vaginal, and is absolutely indicated in contracted and deformed pelves and where for any reason delivery through the natural passages is impossible. It is relatively indicated when the cervix is placed high and where the vagina is undilated, as in primiparæ.

So far as the child is concerned, the results are good; but for the mother, whose interests are paramount, the mortality is too high, owing to lowered resistance, increased shock and infection, to permit of its adoption as a routine method of emptying the uterus in eclampsia.

It is well recognized that statistics can be employed to

justify almost any plan of treatment. Now there is a growing disposition to challange any method of forced delivery, in favor of the expectant plan of treatment, its advocates claiming that sedatives and derivatives, and the deliberate induction of labors will save more patients than any method which involves an anæsthesia with operation, on account of the great susceptibility to shock which these patients have.

Following is a statistical summary of the eclamptic patients treated in this hospital since 1905, that is, since the establishment of the card index system. It is necessarily incomplete because the histories are incomplete.

During this period 41 eclamptic patients were admitted to the hospital, of whom 19 recovered and 21 died. One patient was discharged undelivered. Of these 40 patients:

	Mother.		Child.		Unknown.
	R.	D.	R.	D.	
24 were treated by accouchement forcé	9	15	8	12	4
7 were treated expectantly	4	3	0	2	5
5 were delivered prior to admission .	3	2	0	0	5
4 had no detailed history	3	1	0	0	4
40	19	21	8	14	18

RESULTS OF ACCOUCHEMENT FORCÉ.

	Total.	Mother.		Child.		Unknown.
		R.	D.	R.	D.	
Version	9	3	6	3	6	0
Forceps	9	3	6	2	5	2
Vaginal hysterotomy	2	0	2	1	1	0
Cæsarean section	2	1	1	2	0	0
Cervical dilatation (no details recorded)	2	2	0	0	0	2
	24	9	15	(mortality, 62.5 per cent.)		

The account of the patient who was subjected to the classical Cæsarean operation, and recovered, in a little more detail, is as follows:

Carrie B., aged twenty-nine years, white, Polish, primipara, supposed to be eight and one-half months pregnant, was admitted

to the hospital in a semicomatose condition following convulsions at home. She appeared to be a fat woman, with much swollen and cyanotic face and swollen limbs. Fœtal heart sounds were audible and the head floating. Vaginal examination shows a long nulliparous vagina, cervix high up, long and uneffaced; os closed; systolic blood pressure 210.

Owing to the unfavorable condition for forced dilatation of the birth canal an expectant plan of treatment was adopted. Sixteen ounces of blood were withdrawn from the arm, croton oil was given, and four ounces of a saturated solution of magnesium sulphate was passed into the stomach through a tube; hot packs; nitroglycerine and veratrum were given by hypo.

From her admission at 7 A.M. until 7 P.M. no results had been secured from the efforts at purgation, and during this period she had had eight convulsions. A compound enema now started the bowels and three good motions followed. Six hours later her systolic blood pressure was reduced to 145, and twelve hours later it was 115. She had no more convulsions. Two days later her systolic pressure fluctuated between 122 and 170 at different periods of the day in spite of very active treatment with chloral hydrate, veratrum viride, purging, and sweating. On the third day, it was 195 to 200. On the fourth day it was 200 at 1 A.M., and 238 at 8 P.M. On the fifth day the condition was unchanged and operation was decided upon, the indications for Cæsarean section being primipara, with floating head and undilated cervix high up.

For three or four days after operation the kidney output was very scanty, she remained much swollen, and the blood pressure, which fell to 145 immediately after operation, rose to 192 by noon of the following day and then slowly subsided to about normal. A week after the operation she was well started on the way to recovery, which was uneventful thereafter.

REMARKS ON THE TREATMENT OF PUERPERAL SEPTIC INFECTION AND REPORT OF A CASE OF SEPTIC THROMBOPHLEBITIS OF LONG DURATION¹

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THE treatment of puerperal septic infection in the great majority of cases is, I think I may fairly say, simple and satisfactory. But there is a small percentage of them in which grave surgical operations must be considered. The difficulty in deciding upon the course to pursue in these cases is much increased by the absence of clinical or pathological guides as to the course any given case will follow.

Perhaps most of the cases are due to infection of the endometrium or placental site with putrefactive or mild pyogenic organisms beyond which the morbid process does not extend. These are readily cleared up by the removal of the necrotic and sloughing débris. When, however, the organisms are more virulent and invade the deeper uterine structure, its veins and lymphatics, the problem becomes more complex and its solution more difficult. If the process goes on to abscess formation in the pelvic cellular tissue, a not uncommon termination, the treatment to pursue is obvious, namely, to open the abscess. If there is lymphangitis or thrombophlebitis without suppuration, what shall we do?

¹ Read before the Obstetrical Society of Philadelphia, January 5, 1911. Reprinted from the American Journal of Obstetrics and Diseases of Women and Children, vol. lxiii, No. 4, 1911.

Some of these cases will recover, others (perhaps half of them) will die. If we should quite uniformly remove the pelvic organs for this condition before the morbid process extended beyond them, we should have a fairly large mortality, and many women needlessly deprived of their pelvic organs. In 1902, Trendelenburg successfully operated upon a puerperal case of septic thrombosed veins by ligation of the veins. Successful operations of this kind are also reported by Williams, Freund, Bumm, Vineberg, Seeligman, and others. It is obvious that such an operation, to effect a cure, should be undertaken before the thrombus has extended to the vena cava, or better still to the iliacs. Owing to the severity of these cases and the gravity of operations undertaken for their cure, the great majority of them will be treated by means other than surgical, or at least by trifling surgical measures.

On *a priori* grounds an antitoxic serum should prove very valuable. Inasmuch as the streptococcus is the organism most commonly found in this condition, we would expect an antistreptococcic serum to give results comparable with those of antidiphtheritic serum. In 1899, Williams made an exhaustive report of the cases treated up to that time with serum, and found the mortality no better than in the cases treated without it. Smaller doses were given then than now, and in severe cases it would be proper to give, in addition to other remedies, large and repeated doses of a polyvalent antistreptococcic serum. Begin with 80 c.c. and repeat every six hours until 320 c.c. have been injected. An effort should be made to find the specific organism in the blood or the secretions. But after all the valuable time which such an investigation requires should not be lost by delaying the use of the serum.

A yet more modern treatment of infection is that of bacterial inoculations. In this procedure the actual infecting organism is secured from the blood, or the focus of infection, cultured, sterilized, and suspended in salt solution, thus giving rise to the autogenous vaccine, which is to be administered

by the hypodermic syringe. The dosage is to be regulated by the result which follows. "It must be remembered" (say Deaver, Da Costa, and Pfeiffer) "that a vaccine depends for its action not upon any direct antitoxic or antibacterial substances, but acts only secondarily by exciting the production of these substances by the patient himself;" or to use Wright's conception, "The rationale of vaccine therapy is the exploitation in the interest of the infected tissue of the unexercised immunizing capacities of the unaffected tissues."

"This fact explains at once why no effect is to be expected in one already overwhelmed with infection, and makes it necessary both to use a smaller dose in proportion as the patient is more toxic, and to wait until reaction is established before giving the next dose. The best rule is this: The sicker the patient and the less satisfactory the reaction, the smaller and less frequent should be the dose. It is well to observe that this is the exact opposite to that which holds good for the antitoxic serums."

Efforts to destroy the infecting organisms or their toxins by injecting some antiseptic directly into the circulation have from time to time been made. Of these, the best known is colloidal silver (collargol), which may be used in a 1 per cent. solution and injected directly into a vein. As much as 0.06–0.10 grams (=0.9 to 1½ grains) is recommended by Dimitrin.

I have on several occasions given carbolic acid m_x in a pint of normal salt solution by hypodermoclysis, but am not sure that the good result which has followed was due to anything but the salt solution.

The use of Credé's ointment is based on a similar belief. Fifteen to forty-five grains are to be well rubbed into the skin of the loins or inside of the thighs twice daily.

Nuclein preparations have been administered with a view to increasing phagocytosis. I have employed them, but with how much profit it is difficult to say.

The resisting powers of the patient should be raised and kept at the highest possible level by the use of concentrated

nourishment, such as peptonized milk, eggs, predigested beef, grape juice, junket, broths, etc., given at frequent intervals and in such quantities as the stomach will tolerate.

Tonics and stimulants—iron, arsenic, strychnine, quinine, digitalis, strophanthus, camphor, and whisky were all employed at one time or another during the long illness of the patient whose history I am about to relate—and large quantities of salt solution both under the skin and into the bowel were used and in my judgment were most valuable additions to the therapy.

And last, but not least, I believe that ergot judiciously employed has a distinct prophylactic value. I do not use it routinely after normal labors, but if there be a flabby uterus with offensive lochia and slight elevation of temperature I conceive that the oxytoxic action of the drug will cause the uterus to expel the clots and putrid discharges as well as tighten up the sinuses and thus to a marked degree minimize the absorption of toxic products.

The case which I have to report is as follows:

Mrs. S., aged thirty years, was confined on June 10, 1910. I first saw her nineteen days later. The physician in attendance informed me that the labor had been normal, without the use of instruments, and that the placenta had come away completely. The patient nursed her child. Vaginal douches of creolin solution were given by the nurse and upon the eighth day the patient "felt as if something on the left side had been struck" while the douche was being administered. A few moments later a very painful chill followed. The next day douching was repeated without ill effect. Three days later she had another chill. She got out of bed the following day, but pain in the left groin was so severe that she sent for the doctor, who advised her to go back to bed, and treated her with douches and an ice cap to the head and groin for a week, when I first saw her and had her admitted to the Episcopal Hospital. Her history showed that she was a healthy, hard-working woman. She had had the usual diseases of childhood, but had never had typhoid fever, malaria, rheumatism, cardiac or renal disease, nor pelvic disease of any kind. She was married at eighteen, had four children living and well, and no

miscarriages. On admission to the hospital, examination showed the patient to be pale, with vagina and cervix postpuerperal and without notable laceration. The size of the uterus was about normal (perhaps a trifle enlarged) for the date of the puerperium and there was marked tenderness on the left side of the uterus and in the left groin. No distinct mass was palpable but rather a sense of thickening and impaired mobility. Temperature 103° . On the following day at noon it was 100° and at 4 P.M. 103.8° , pulse 104 and respiration 40. She passed 24 ounces of urine. The temperature remained for four days between 100° and 104° and pulse between 90 and 100. On July 6, that is eight days after admission to the hospital, the patient had her first chill since she came into the hospital. It occurred at 5.30 A.M. and lasted twenty minutes. Two days later at 2 A. M. she had another chill, three days later at 1 A.M. a third, two days later at 7 P.M. another. Again in two days at 10 P.M. another and so on, the chills occurring mostly at night.

The temperature in the meanwhile fluctuated between 97.6° and 104.6° , but did not go high every day. The pulse varied between 72 and 100, mostly in the 80's. On the fortieth day the temperature after a chill was 105° and now the daily temperature curve was of a more regular form— 100° to 103° . On the forty-eighth to fiftieth days, *i. e.*, two or three days after the injection of a vaccine made from organisms taken from the endometrium, the temperature covered the remarkable range of nearly 9° , *i. e.*, from 95.2° to 103.8° . Then it continued to fluctuate pretty evenly until the seventieth day when it remained about normal for one day. The patient's condition except for the very marked anæmia (one blood examination showed red blood corpuscles 1,800,000, white blood corpuscles 10,000 and hæmoglobin 24 per cent.) seemed far better than the prolonged fever would indicate. Much of the time she had a good appetite, complained of nothing whatever, and as she lay in bed with sometimes a hectic flush, she had the appearance of such perfect health that it was incredible that she should be the host of so relentless an infection. Slight tenderness remained in the left fornix of the vagina and left groin, but the womb was well contracted. At no time was there palpable a large, distinct or fluctuating mass behind the cervix, but only a slight thickening and tenderness to the left of it. On August 18, seventy days after labor, following a period of relative comfort with a temperature fluctuating from normal to $101^{\circ}+$, and pulse in the 80's and 90's, the patient was

somewhat restless in the evening, and early the following morning awoke with a severe cramp in the right leg, vomited, and shortly afterward, almost simultaneously, got a pain in the left leg. The pain was very severe, the legs became swollen, cyanotic, and cold. The vomiting continued, the patient was covered with perspiration, the pupils widely dilated, the pulse very rapid and feeble, almost imperceptible, the heart sounds were weak, the first and second being very much alike. There was great restlessness, thirst, and nausea; and it was thought by the doctors who saw her from time to time throughout the day that she could not live more than a couple of hours. From this time medication and food had to be administered by hypodermic and by the bowel, the nutritive enemas being continued for about two weeks when nourishment by the mouth was gradually resumed. The temperature continued to fluctuate between 97 and 103° until the eighty-seventh day when it remained near the normal line with one or two slight exceptions until the one hundred and second day when she passed out of my immediate care at the hospital. By this time she had lost much flesh, had a septic diarrhoea with stools and urine passed involuntarily, and a small bed sore over the process of an upper dorsal vertebra. After she returned to her home, these conditions grew worse; the stools increased to twelve to fifteen daily and numerous pustules developed on the back. She became more emaciated, less able to take nourishment, and became mildly delirious a few days before her death, which occurred on the one hundred and fourteenth day after the birth of the baby.

No autopsy was had, but I regarded the case as one of septic thrombophlebitis, beginning in the veins of the left broad ligament, extending up into the iliac and finally occluding the common iliacs at their junction. With this diagnosis in mind I considered the matter of opening the abdomen and ligating and removing the thrombosed vessels as has been done by Trendelenburg, Freund, Bumm, Williams and others, but did not adopt the plan because of the length of time which had elapsed before she came under my observation, and because I could not tell how far the thrombus had advanced. Numerous blood cultures were made with negative results. A culture from the intra-uterine cavity yielded a diplo-streptococcus from which a vaccine was prepared and administered without apparent result, except it be credited with the marked temperature range above referred to, which followed about three days after the first dose.

Following is a record of the numerous blood examinations:

July 1, 1910.—W. C. B. 18,800.

July 5, 1910.—Blood culture negative.

July 7, 1910.—R. B. C. 3,180,000. W. B. C. 16,000. Hb. 45 per cent. Widal reaction negative.

July 12, 1910.—W. B. C. 23,200.

July 17, 1910.—Blood culture negative. Poly. 80. Lymph. 15. Mono., 3. Trans., 1. Eosin 1. R. B. C. 3,550,000. W. B. C. 15,760. Hb. 42. Malarial inspection negative. Intra-uterine culture showed diplo-streptococcus.

July 23, 1910.—Vaccine prepared from intra-uterine culture given. W. B. C. 19,440 before vaccine was given.

July 24, 1910.—W. B. C. 18,080 after vaccine was given.

July 26, 1910.—W. B. C. 16,240.

July 30, 1910.—W. B. C. 16,000.

July 31, 1910.—W. B. C. 17,000. Blood culture negative. Poly. 86. Lymph. 7. Mono. 1. Trans. 6.

August 2, 1910.—W. B. C. 9360.

August 3, 1910.—W. B. C. 10,000.

August 5, 1910.—R. B. C. 1,800,000. W. B. C. 10,000. Hb. 24.

August 6, 1910.—Blood culture negative. R. B. C. 2,090,000. W. B. C. 10,000.

August 8, 1910.—W. B. C. 14,000.

August 11, 1910.—R. B. C. 2,790,000. W. B. C. 11,000. Hb. 30.

August 12, 1910.—Iodophilia negative. Basophilia negative. Blood cultures negative.

August 26, 1910.—Blood culture negative.

September 10, 1910.—Urine straw color, faintly acid. Alb., heavy trace. No sugar, no casts, triple phosphates. Many W. B. C. No R. B. C. Many bacteria.

TWO CASES OF RUPTURED EXTRA-UTERINE PREGNANCY, ONE OF THEM IN AN INSTANCE OF UTERUS BICORNIS ¹

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I HAVE thought that it might be of interest to report these two cases which have recently occurred in my service, not only because in one of them an interesting anomalous condition was found, but because they both illustrate the uncertainty, and even the difficulty sometimes experienced in making a diagnosis of the lesion from which both suffered.

CASE I.—Ruptured extra-uterine pregnancy in a case of uterus bicornis; operation; recovery.

This might more properly be termed rupture of one cornu, due to pregnancy in a uterus bicornis.

The patient, A., aged nineteen years, married, was admitted from the Receiving Ward, with the diagnosis of appendicitis, on February 16, 1912.

According to the case notes, she had been feeling badly for the past three weeks, and had had frequent attacks of pain in the right lower abdomen. She had not been pregnant before, and had always menstruated regularly, the last time, however, having been in December. Her January period was missed. Four days previous to admission she began having severe pain on the right side of the abdomen, vomited several times, and is said to have fainted.

Her appearance was markedly anæmic. She was of very fair complexion, however, and both she and her relatives stated that she had had little color for a long time.

¹ Reported at a meeting of the Episcopal Hospital Clinical Society, March 18, 1912.

On examination, the abdomen was found slightly distended, and there was muscular rigidity of the right lower segment, which was tender on palpation and dull on percussion. An ill-defined mass the size of one's fist had its centre at McBurney's point.

The breasts were negative. On vaginal examination the cervix was found slightly softened, and from the os there was a very slightly blood-tinged discharge.

The pulse, on admission, was 130, the temperature 97° . The blood examination gave the following result: Hæmoglobin, 46 per cent.; white blood corpuscles, 31,600; polynuclears, $89\frac{1}{3}$; mononuclears, $\frac{1}{3}$; transitionals, 2; lymphocytes, $8\frac{1}{3}$.

The indications pointing to a marked septic infection, with some uncertainty as to the exact abdominal lesion, and the generally unfavorable condition of the patient for immediate operation caused me to decide to pursue an expectant course, and accordingly stimulating treatment and enteroclysis were ordered.

On the ensuing days the following observations were noted:

February 17, highest temperature, $99\frac{3}{5}^{\circ}$, highest pulse rate 148. Blood examination: hæmoglobin, 42 per cent.; white blood corpuscles, 18,640; polynuclears, 86 per cent.; mononuclears, 0; transitionals, 4; lymphocytes, 10.

February 18, highest temperature, 101° ; highest pulse, 148.

On February 19, the temperature ranged from $99\frac{3}{5}^{\circ}$ to 103° , and the pulse from 128 to 132.

On February 20, the highest temperature was $100\frac{2}{5}^{\circ}$; the pulse varying between 112 and 132. Blood examination: Hæmoglobin, 42 per cent; white blood corpuscles, 14,400; polynuclears, 83 per cent; mononuclears, 0; transitionals, $3\frac{1}{2}$; lymphocytes, 12.

On February 21, the temperature rose to $102\frac{4}{5}^{\circ}$, while the pulse rate was from 120 to 132.

On February 22, the temperature, which was $101\frac{4}{5}^{\circ}$ at 4 A.M., fell to $99\frac{1}{5}^{\circ}$ at 8 A.M., and did not rise again above 100° . The pulse remained about the same as on the previous day.

On February 23, the temperature varied from $98\frac{4}{5}^{\circ}$ to $99\frac{4}{5}^{\circ}$; and the pulse from 96 to 104. Examination of the blood showed the hæmoglobin to be 45 per cent., while the leukocytic count was the same as on February 20.

While marked improvement in the patient's general condition had now taken place, the local conditions remained practically unchanged.

I operated upon her on February 24, opening the abdomen through the right rectus muscle. Some fluid and a considerable

amount of old coagulated blood were found in the peritoneal cavity. The omentum, greatly thickened, reached down into the pelvis and was adherent to the fundus and right side of the uterus. The mass, mentioned above as being central in the region of McBurney's point, was found, on separating the omentum from its pelvic adhesions, to consist of partly organized blood clot. This, with the thickened portion of the omentum to which it was adherent, was removed. The pelvis contained some blood clots which were also undergoing organization. I naturally expected to find a ruptured tubal pregnancy. On examining the parts, however, I discovered that the patient had a uterus bicornis, and that what had happened was that rupture of the right, and much smaller horn, in which pregnancy had taken place, was the lesion. The rent had occurred on the posterior aspect, the cavity was filled with clot, and no bleeding was now taking place. The right tube was continuous with the right cornu, and both it and the ovary were normal. The bicornuate condition of the uterus was not complete. The division extended only for a distance of an inch and three-quarters or two inches from the fundus. Between the right cornu and the body of the womb was a thin bridge of tissue.

Applying a clamp close against the main portion of the uterus, and passing a ligature through the broad ligament, I severed the ruptured right cornu, with its tube and ovary. The wound of the uterus was closed with a continuous chromicized catgut suture, which on removal of the clamp was found to control hemorrhage perfectly. A second row of interrupted sutures of the same material was applied to cover over the raw edges.

The foetus, which was evidently of about two months, was found in the right loin to the outer side of the ascending colon, just above the caecum.

Before closing the abdomen, I inserted into the pelvis a glass drainage tube and a Mikulicz drain.

The course of the patient after operation was excellent, there being no interruption in her recovery, with the exception of a stitch abscess, which somewhat delayed the healing of the wound. She regained her strength gradually, and was discharged in good condition on April 16.

The specimens, which I exhibit, show the ruptured cornu with its normal Fallopian tube and corresponding ovary, the foetus, and the clot mass and thickened omentum which,

because of the location of the mass, at the first inspection of the patient on her admission to the hospital, together with the leukocytic and differential count, led to the provisional diagnosis of appendicitis with abscess.

CASE II.—Ruptured tubal pregnancy; operation; recovery. R., aged twenty-four years, married, admitted to the Receiving Ward on February 26, 1912, with the diagnosis of salpingitis.

Her history was that she had been married for seven years, but had had no children. Menstruation had been regular, but she had missed the last two periods. Until recently she had had no leucorrhœal discharge.

Her illness began one week before admission, when she experienced a sudden, acute pain in the right tuboövarian region. This was followed by vomiting, which was repeated during the next day or two.

The patient was a healthy, well-developed looking woman, with excellent color.

She complained of pain in the right inguinal region of the abdomen, which was tender on palpation. Rigidity of the muscles was not marked.

Vaginal examination revealed a whitish yellow discharge, the uterus slightly enlarged, a mass occupying the left tubal region, and a smaller one, very tender to pressure, on the right.

Her temperature on admission was $99\frac{3}{5}^{\circ}$, her pulse 112. That evening the temperature was 100° , the pulse 96.

The next day, February 27, the temperature varied from $98\frac{4}{5}^{\circ}$ in the morning to $99\frac{4}{5}^{\circ}$ in the evening, and the pulse 80 to 88. The leukocytic count 10,320.

The indications pointing to acute salpingitis, the patient was kept in bed, an ice bag was placed over the region of tenderness, and vaginal douches ordered. Within forty-eight hours the symptoms were much less acute, and the patient was doing well. A week later she was feeling better, but still had some pain, although not severe, in the region of the right adnexa. The temperature was running between normal and 99° , or a little above, and the pulse from 80 to 90. On March 9, the leukocytic count was 11,520. On March 13, acute tenderness having subsided, I advised operation, which was done the next day.

On opening the abdomen, the omentum, which was thickened at this part, was found extending into the pelvis, and enveloping and adherent to the right Fallopian tube, which was the seat of a

tubal abortion. On freeing the omentum the rupture of the tube was disclosed, the amniotic sac was broken and a six to eight months' foetus escaped, leaving the cavity partly filled with blood clots. Adhesions had formed between the enlarged tube and the peritoneum of the right pelvic wall. The mass was then freed, and the tube tied off with silk and removed. The abdomen was closed without drainage.

The patient has done well after operation, save for a rise of temperature to $101\frac{1}{5}^{\circ}$ at the end of the first twenty-four hours. She was out of bed in two weeks, and was discharged on April 6.

The specimens show the seat of pregnancy in the tube, the rupture, and the fetus. So thoroughly was the rupture of the tube covered and sealed by the adherent omentum that no free hemorrhage had occurred.

The chief interest in these cases, aside from the anomaly presented by one of them, lies in the question of diagnosis. While a ruptured ectopic pregnancy with sudden, free hemorrhage, is not difficult to recognize, the cases I now report, as their histories show, offered difficulties as to their correct interpretation. In the case of the first patient, the length of duration of her illness, the high leukocytic count, and the high polynuclear percentage, pointed, as I have already remarked, to a septic condition, and the mass in the right lower quadrant of the abdomen led one to suspect an appendicular abscess, although I entertained the possibility of the diagnosis which proved to be the correct one. It is true that she had missed two menstrual periods, and that she was markedly anæmic; but her pallor was said to be not abnormal.

In the other case the leukocytic count, the bilateral involvement of the Fallopian tubes, as shown by vaginal examination, and the absence of any indication whatever of hemorrhage, seemed to justify the diagnosis of acute salpingitis, the missed menstrual periods suggesting the probability, under the circumstances, of normal pregnancy.

VACCINATION AND ITS DISCOVERER¹

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To speak of Jenner and vaccination to medical men is like "carrying coals to Newcastle." Nevertheless, I think it is well from time to time to reacquaint ourselves with the great names in medicine; and no name is greater than that of Jenner in the realm of preventive medicine.

Edward Jenner was born May 14, 1749, at Berkeley, sixteen miles from the Episcopal city of Gloucester. His father was the Rev. Stephen Jenner, rector of Rochampton and Vicar of Berkeley. His mother was the daughter of the Rev. H. Head, a former vicar of Berkeley. His first school was at Wooten-under-Edge, where he was under the care of the Rev. Mr. Clissold. From there he was removed to the Rev. Dr. Washbourn at Cirencester.

Jenner's school career was a short one. At the age of thirteen he began his professional education under Mr. Daniel Ludlow, of Sudbury. From there he entered as a student at St. George's Hospital, where his name appears in the list of students from 1770; and when he was twenty-one he went as house-pupil to the great John Hunter. Nothing could have been more fortunate for one who had an innate love for natural history than that he should come under an influence such as Hunter's. The young pupil came with a fair knowledge of both zoölogy and geology; the fossiliferous rocks of his native county had given him ample oppor-

¹ Read before the Episcopal Hospital Clinical Society, May 20, 1912.

tunity to study geology and the collecting of fossils was a hobby that he retained throughout life. To the young man with these tastes Hunter's house with its menagerie and collection of specimens must have been a paradise. His love for natural history nearly robbed medical science of Jenner's discovery. He arranged to prepare the specimens brought home by Captain Cook in 1771, and was offered the post of naturalist to the next expedition, which sailed in the following year. This offer he declined and settled down to country practice in his native town of Berkeley.

Here for some years he led the quiet life of a country doctor, with ample leisure for his natural history pursuits. Baron, in his life of Jenner, gives us a picture of him from the pen of his great friend, Edward Gardner of Frampton. He says: "His height was rather under the middle size; his person was robust but active and well formed. In his dress, he was particularly neat and everything about him showed the man intent and serious, and well prepared to meet the duties of his calling. When I (Baron) first saw him, it was on Frampton Green. I was somewhat his junior in years and had heard so much of Mr. Jenner of Berkeley that I had no small curiosity to see him. He was dressed in a blue coat with yellow buttons, buckskins well polished, jockey boots with handsome silver spurs, and he carried a smart whip with a silver handle. His hair, after the fashion of the times, was done up in a club and he wore a broad-brimmed hat."

Baron's description of his first interview with Jenner is as follows: "The simple dignity of his aspect, the kind and familiar tone of his language and the perfect sincerity and good will manifested in all he said and did, could not fail to win the heart of anyone not insensible to such qualities. He was dressed in a blue coat, white waistcoat, nankeen breeches, and white stockings." We are grateful to him who told us that Milton wore large buckles and that George Washington broke in his own horses, and at some future day the curious reader may be thankful for particulars descriptive

of Jenner's habits. It is from such contemporary descriptions only that pictures of the great men of the past can be completed. For this reason it is worth quoting a paragraph from the obituary of Jenner in the *Gentlemen's Magazine*, volume xciii, page 104, which evidently was written by some one acquainted with him: "In his housekeeping nothing was gaudy but everything was good. The cookery was tastefully and fashionably set out; the wines, commonly five or six kinds, old and of fine flavor. At a strikingly innocent trait of character, the philosopher as a keen observer would smile cheerfully, but the writer of this never saw him indulge in what is called 'a horse laugh.' "

In 1778, Jenner was crossed in love. This he took very much to heart and his correspondence with Hunter did not improve matters. Hunter had evidently heard a false report as regards Jenner, for he writes, "I was told the other day that you were married and to a lady of fortune. I hope it is true for I do not know of anyone more deserving of one." On hearing the truth, Hunter again writes, "I own I was at a loss to account for your silence and I am sorry for the cause. I can easily conceive how you must feel, for you have two passions to cope with, viz., that of being disappointed in love and that of being defeated; but both will soon wear out, perhaps the first soonest. I own I was glad when I heard you were married to a lady of fortune, but let her go. Never mind her. I shall employ you with hedge-hogs for I do not know how far I may trust mine."

Hunter's prophecy that Jenner's disappointment in love would wear out, came true, for ten years later, in 1788, Jenner was married to Miss Catharine Kingscote, and although Mrs. Jenner was of delicate health, she did much for the poor and suffering in her neighborhood. In this her efforts were ably assisted by her husband. Jenner took an active part in all local work; he was Justice of the Peace for the County of Gloucester and also filled the office of Mayor of Berkeley.

There were three children born to Jenner. John Hunter stood godfather to the eldest born. The following is his reply to Jenner's request that he would undertake that office:

"JANUARY, 1789.

"DEAR JENNER:

"I wish you joy. It never rains but it pours. Rather than the brat should not be a Christian, I will stand godfather, for I should be unhappy if the poor little thing should go to the devil because I would not stand godfather. I hope Mrs. Jenner is well and that you begin to look grave now that you are a father.

Yours sincerely,

JOHN HUNTER."

The vicarage at Berkeley where Jenner was born is no longer in existence, but the Temple of Vaccinia is still standing. The hide of the cow from which Sarah Nelmes was infected is now in the Curator's room at St. George's Hospital. An inscription states that it was presented to the hospital on October 14, 1857, by Jenner's son.

The year 1796, was a memorable one in Jenner's history, as on May 14 of that year he performed his first inoculation with cowpox. The subject was a boy, aged eight years, named John Phipps, and the matter was taken from the hand of Sarah Nelmes, a dairy maid, who had become infected by her master's cows. This was an anxious time for Jenner, for on July 1 smallpox matter taken directly from a pustule was inserted but no disease followed. At once he wrote off to his friend Gardner, to tell him of his success. After describing the inoculation, he proceeded: "Having never seen the disease but in its casual way before, that is, when communicated from the cow to the hand of the milker, I was astonished at the close resemblance of the pustule in some of their stages to the smallpox pustules. But now listen to the most delightful part of my story: The boy has since been inoculated with smallpox, which as I ventured produced no effect. I shall now pursue my experiments with redoubled ardor."

This subject seems first to have attracted Jenner when he was a pupil at Sudbury. A young girl came there for advice, and on smallpox being mentioned, she exclaimed, "I cannot take that disease for I have had cowpox." During Jenner's pupilage he mentioned the matter to Hunter, who does not seem to have been much struck with the idea, but he gave his pupil this good bit of advice: "Do not think, but try. Be patient; be accurate." On Jenner's return to Berkeley the idea was constantly in his mind. He found that the opinion of the young girl at Sudbury was a general one among the milkers in and around Berkeley. To get at the truth of this opinion was his great object, but it was not until 1780 that he felt sufficient confidence in his conclusions to warrant his imparting them to others.

One hundred and sixteen years have passed away since Jenner's first successful vaccination on May 14, 1796. His brilliant idea, however, which he pondered for more than twenty-five years, was that smallpox might be abolished by the universal adoption of vaccination. Others had vaccinated before Jenner, but he was the first to rouse the civilized world to take an active interest in the subject; and we must not forget that vaccination was not the outcome of laboratory experiment but a practice resting upon a common experience in many countries of Europe (not to mention Mexico and Persia), that dairymaids and others who had sore hands from milking cows affected with cowpox were afterwards found to be protected against smallpox.

It was hardly to be expected that Jenner's discovery would escape the odium theologicum. Many sermons were preached to show the wickedness of vaccination and one preacher went so far as to try to demonstrate that the cowpox vaccination was anti-Christ.

The time at my disposal will not permit any extensive review of the history of vaccination, but I would like to refer to two historic instances and one of more recent date:

The first was in 1889. The deaths from smallpox in six

provinces of Spain under optional vaccination were 11,050. The death-rate from smallpox in Germany for the same year under compulsory vaccination was 4.

The second instance is that of Sheffield where during the epidemic of 1887-1888 the whole population was examined as to the vaccination condition, and note again the amazing contrast between the fatality among the unvaccinated and that among the vaccinated. Of 268,397 persons returned as vaccinated, only 1.55 per cent. were attacked by smallpox and .007 per cent. died; while of 5715 persons returned as unvaccinated 9.7 per cent. were attacked and 4.8 per cent. died. Furthermore, of some 21,000 persons who were re-vaccinated not one took the smallpox.

The modern instance is reported by Victor G. Heiser, Chief Director of Health in the Philippine Islands. During the Spanish times it was necessary each year during the dry season to erect in Manila a large temporary hospital to which many hundreds of victims of smallpox could be taken. Great numbers of them died. During the past five years not one person who had been successfully vaccinated has died from smallpox, nor has anyone died in Manila from smallpox since 1909. Since 1907, when systematic vaccination was completed, of the six provinces near Manila which have a population of over one million and which from time immemorial had an annual death-rate of at least six thousand persons, not one person has died who had been successfully vaccinated and only a few scattered cases have occurred.

To these three instances I could add many others of similar tenor; but they are enough to convince any unprejudiced person that the diminution of smallpox in any country is not due to improved sanitation but to vaccination. The Sheffield instance proves it beyond doubt or cavil, for here the sanitary conditions were uniform and should affect all persons alike; but we see the most striking difference between the vaccinated and the unvaccinated. And furthermore, no one will claim that the Philippines have suddenly

jumped into an ideal sanitary condition since the American occupation in 1907. What then has caused the striking diminution in smallpox in these islands, if not vaccination?

In view of all this, does it not seem strange that we know so little of him whose patient labors have prevented so much suffering, desolation, and death? The memory of Edward Jenner and the one-hundredth anniversary of his great discovery were honored in many countries with public celebrations of one kind or another. In France, Germany, Russia, and the United States glowing tribute was paid to the memory of Jenner and his inestimable discovery was proclaimed to be the grandest conquest of medical science.

Jenner's predecessors were the milkmaids of Gloucestershire and elsewhere who had found out for themselves that cowpox was a safeguard against smallpox, before the insight and painstaking labor of Jenner transformed a piece of folklore into a scientific truth and made the medical profession all over the world receive it as such. It is also unquestionable that he was not the first to inoculate cowpox with the object of giving protection against smallpox. The first vaccinations of which there are any record were performed by Benjamin Jesty, a farmer of Yoeminster in Dorsetshire, who in 1774 inoculated his wife and two children, aged respectively two and three years. He, himself, had had cowpox before by contagion from the cow. Jesty's vaccinations were successful and his family remained proof against smallpox inoculation and contagion.

Again, in 1791, a Holsteiner named Peter Plett successfully vaccinated some children. He was tutor in a family at Schonwaide in Holstein where the milkmaids had also discovered the protective efficacy of cowpox against what they called "the most serious disease." Plett having seen a medical man perform variolous inoculations, conceived the idea of inoculation by cowpox. Accordingly, he vaccinated his employer's three children, making incisions with a pocket-knife on the

back of the hand, between the thumb and forefinger. This rough-and-ready method of vaccination was successful and when an outbreak of smallpox occurred in the place, three years later, the children of Plett's employer were the only ones spared by the disease.

Neither Plett nor Jesty, however, appear to have followed up the matter, hence nothing came of their happy thought. Let Jesty and Plett have the credit that is their due; they had a glimpse of the truth but they passed away and left no trace. To Jenner, alone, belongs the glory of having given to mankind the means of ridding themselves of a hideous scourge and to science a new truth which, fruitful as it has already been, contains within it the germs of development yet undreamed of. If in these days the fear of smallpox has become, for those who are content to be guided by the logic of facts, almost as remote as the fear of leprosy, we owe our deliverance to Jenner. Were it not for the safeguard with which he has provided us, it is scarcely too much to say that, with the present ease and rapidity of communication between the most distant parts of the earth, the human race would be in constant danger of decimation by smallpox. As for the fools and fanatics who decry Jenner and seek to undo his work, it is only charitable to believe that "they know not what they do."

In a striking passage Sir John Simon says: "It is a favorite reflection among philosophers that if departed great benefactors of our race could now and then look down on the harvest fields where mankind, age after age, are gladdened by the fruits of their labor, they would, in general, find themselves less remembered than perhaps their terrestrial ambitions had desired." Doubtless, this is true; but let the noble compensation be noted, that often the thoroughness of the reformer's victory is that which most makes silence of the reformer's fame—for how can men be adequately thankful for redemptions when they have no present easy standard, no contrast between yesterday and today, by which to measure the greatness

of them. In some cases ignorance best tells its tale by swaggering against the truths which protect it. At the antivaccination meetings, of which we now occasionally read, where some pragmatical quack pretends to be making mincemeat of Jenner, how small would become the voice of the orator and how abruptly would the meeting dissolve itself, if for a moment the leash were removed with which Jenner's genius holds back the pestilence, and smallpox could start into form before the meeting as our grandfathers saw it but a century ago.

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